THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—27TH YEAR. SYDNEY, SATURDAY, JUNE 8, 1940. No. 23.

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STUDIES IN TUBERCULOSIS. II: TUBERCULOSIS INFECTION IN NURSES, WITH SPECIAL REFERENCE TO INCIDENCE IN NEW SOUTH WALES.[1]

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THE newer knowledge of the natural history of tuberculosis, which has been current since the discoveries of Koch half a century ago, and the development of the cutaneous tuberculin test by von Pirquet thirty years ago, opened the way to a great volume of epidemiological study all over the world. Great differences have been demonstrated in the incidence and distribution of tuberculosis infection between different communities; differences and trends have been observed also in the degree of infection in the same communities over the years; populations have become less densely infected; infection is being first acquired later in life. Theories of the pathogenesis of tuberculosis have had to be broadened.

In the past ten years there has been a great concentration of study on the epidemiology of tuberculosis in closed communities exposed to preternatural hazards of contagion and liability to disease, and of these communities especially the tuberculous household and the hospital workers who come closely in contact with the sick and often with the unrecognized tuberculous patient.

In the communities of nurses and medical students it has been possible to observe that many, contrary to former belief, enter without having been previously infected with tuberculosis. It has been possible to follow the progress of the entrants, to

[1] Work carried out with the aid of a grant from the National Health and Medical Research Council of the Commonwealth of Australia. From the Royal North Shore Hospital of Sydney Institute of Medical Research. Submitted as a supporting paper with a thesis for the degree of Doctor of Medicine in the University of Sydney, January 7, 1939.

observe infection occurring in a majority and to note that of those who are infected for the first time in adult life, some soon develop manifest tuberculous disease and others after a longer interval.[1] It has also been shown beyond doubt that those who had acquired their first infection and withstood it before entering the community, very seldom became diseased. With so plain a perspective of the process of contagion leading to infection and of infection to disease, it was natural that many observers were led at once to seek means to interrupt the process at some stage in order to prevent disease. But, in the devising of means to attain the end, confusion of thought, division of opinion and divergence of practice have persisted to the present day, sometimes leading to conflict between outstanding workers inspired with the same ideal. For instance, Geer[2] has written:

> Heimbeck's use of B.C.G. for his non-reacting nurses is hardly warranted as yet; furthermore it is the wrong approach. Institutions can and should be made safe from the standpoint of contagion.

If it is not yet practicable to lay down definitive means for the protection of hospital workers from tuberculosis, it is possible and helpful to set out the approaches to the problem briefly and logically, with especial reference to local conditions, to examine its scope, and to point out the gaps in our knowledge and the means of making them good.

Such an exposition is here attempted, and an investigation is described which has been carried out to determine the incidence of tuberculosis infection in hospital nurses in New South Wales.

The Various Approaches to the Prophylaxis of Tuberculosis in Hospital Workers and their Objections.

The approaches to the prophylaxis of tuberculosis are three, which is the number of links in the chain of contagion, infection and disease. We may aim to snap any one or more of these links.

The Prevention of Contagion.

We may aim to prevent contagion. The tuberculous may be segregated, but this, as Geer admits, involves routine radiography of the chest of all patients entering all hospitals. The objections to this have been those of cost and administration. Costs could be reduced if only those reacting to tuberculin were examined by X rays, but the administration would be clumsier.

Where thorough segregation is impracticable, a rigid anti-contagious technique may be adopted in dealing with all known tuberculous patients, just as in tuberculosis wards. The objections to this are that the tuberculous patient in the general ward would be distressed by receiving special treatment suggesting to everyone that he was a source of danger, that the great emphasis laid on the presence of contagion in the ward might create apprehension among the other patients and unnecessary dread among the nurses, and that the best practicable anti-contagious technique would only lessen and not eliminate the possibility of contagion in the general hospital.

The Prevention of Infection.

Secondly, where the possibility of contagion exists, we may aim to prevent the infection that may lead to disease.

Hospital workers may be recruited only from among those who are unfavourable soil for the tubercle bacillus; or, alternatively, only those who are unfavourable soil may be allowed to come in contact with known sufferers from tuberculosis. Those constituting unfavourable soil are usually said to be, firstly, those over thirty years of age (but it may well be that in the future there will be demonstrated a greater proportion of original infections leading to manifest disease in older persons, just as recently in young adults); and, secondly, those who react to the cutaneous tuberculin test, but who present no clinical or radiographic evidence of tuberculosis, and who presumably have developed resistance to tuberculous disease over and above that which they may have naturally possessed. It has now been shown fairly conclusively[1][3][4] that the last-mentioned reactors are very much less prone to develop tuberculous disease than non-reactors living in the same environment. It may be objected that this plan would involve discrimination between nurses in the allotment of duties, stigmatize certain nurses (with doubtful justification in any individual case) with the allegation of their special susceptibility to develop tuberculosis, and frequently cause acute difficulty in staffing the wards, which difficulty may well be progressive.

Uninfected nurses may be deliberately infected with an attenuated tubercle bacillus, which will not produce disease, but which will mobilize their resistance against subsequent infection with virulent bacilli. This has already been done to sanatorium nurses in European countries which are heavily infected by tuberculosis[1] and in America. To this it may be objected that such treatment would certainly be difficult to introduce in this country, and, if insisted upon, might accentuate the present shortage of nurses.

The general objection usually raised to all these methods of preventing contagion and infection is that infection, if it occurs, does not result in disease sufficiently often to warrant the introduction of methods to which there are the particular objections stated. Much therefore hinges upon the validity or otherwise of this general objection.

The Prevention of Serious Disease.

The methods of attempting to prevent contagion and infection have been discussed. Lastly, we may try to prevent serious disease from developing after infection has occurred. It is to this end that the energies of hospitals are chiefly directed in this country (where any energies at all are directed to the prophylaxis of tuberculosis amongst their staffs). In many institutions the plan of campaign is haphazard or illogically developed or only partly developed, and many would be energetic were guidance forthcoming or facilities available. The steps

of a model scheme are here set out: it is to be understood that its purpose is not the prevention of contagion or infection, but only the timely recognition of disease should it develop after infection has occurred.

First, at the time of entrance to the hospital, a note is made in the course of the medical examination of any family history of bronchitis, asthma or other chest trouble. Those who give such a history are marked for special observation, as will be represented later. There is no doubt that a family history of tuberculosis is frequently suppressed if not sought with tact and if there is not good *rapport* between examiner and examinee. The weight is recorded during the medical examination and from time to time afterwards.

Secondly, each nurse is submitted to a radiographic examination at the time of entrance to the hospital.

Thirdly, each nurse also receives an intracutaneous tuberculin test at the time of entrance.

The weight of evidence is that those who react to the tuberculin test and are radiographically free of parenchymatous pulmonary tuberculosis, rarely develop tuberculous disease subsequently in sanatoria and scarcely ever in general hospitals. Still, those who on entrance give a history of recent continuous exposure to infection in the family or elsewhere, should have the radiographic examination repeated annually or at once should unexplained ill health occur.

Those who do not react to the tuberculin test should be retested every six months. Should a reaction develop, further radiological examination is indicated and should be repeated annually, or more frequently if the reaction was a strong one.

Observation does not cease with the conclusion of the training period, but facilities are made available for its continuance for several years afterwards. After the lapse of several years, according to Heimbeck,[5] the incidence of tuberculous disease in graduate nurses is no greater than in the population at large.

Nurses are encouraged to report ailments to the proper quarter, and the competent authority whose duty it is to examine them, should not appear to be irritated when minor ailments are reported. It was a widespread impression at one large hospital that most nurses would rather carry on with a slight fever than report sick to an ungracious authority.

The food, living conditions, hours and conditions of work and curriculum should be fixed by authority and adhered to.

The Gaps in Our Knowledge of the Incidence of Tuberculous Infection and Disease in Nurses.

In this country there has hitherto been no knowledge of the incidence of tuberculosis infection in nurses, and there has also been a conflict of evidence as to the incidence of tuberculous disease. What little evidence is available has been summarized by Storey.[7] Clarification of the position is made very difficult, for some hospitals with tuberculosis wards, where the incidence of tuberculosis in nurses is known positively to attending physicians to have been very high, will not publish statistics, possibly for fear of causing an even greater shortage of nurses than at present. On the other hand, at the Royal Prince Alfred Hospital, Sydney, where there were 300 nurses in training for a four-year period and where tuberculous patients are admitted, there were only six known cases amongst the nurses in fifteen years. But the routine X-ray examination of nurses was introduced only towards the end of the observation period. At the Royal North Shore Hospital of Sydney the nurses have lived under excellent conditions since 1930, since when, of 200 nurses, only one developed tuberculosis; but here again routine radiographic examination is an innovation, and it is not known whether any broke down after the completion of training.

At another hospital, where it was reported to the directors that only a certain number of nurses had developed tuberculosis in a certain period, one of the physicians remarked that he had more than that number of ex-nurses under his own care suffering from tuberculosis. Still, in the presence of a small known incidence of tuberculosis, and until more definite evidence is forthcoming of undue prevalence in their nurses, hospital authorities cannot be expected to regard the problem as a serious one.

Are Nurses More Susceptible to Tuberculous Infection and Disease than Other Young Women?

Our knowledge of the incidence of tuberculous disease in nurses as compared with the incidence in young women of the same age in other occupations is also as yet very imperfect, though very valuable data have just been published by Heimbeck.[5]

In England (where attention was directed to the question as long ago as 1903 by Theodore Williams) statistics from the Registrar-General have, according to Edwards,[6] only lately been promised and are not yet published. In Australia information is only now being collected (by the National Health and Medical Research Council). From other countries the reports published concern individual hospitals for the most part, and these have been summarized by Edwards.[6]

It is of more value to know how many of this or that group of nurses became tuberculous than how many of this or that group of sufferers were nurses. In this country, where nurses are still recruited largely from the middle classes, a relatively small proportion of those developing tuberculosis would find their way into the large sanatoria.

Original Studies in the Incidence of Tuberculous Infection in Nurses in New South Wales.

In order that information might be gained as to the proportion of nurses in general hospitals in New South Wales who become infected (as opposed to invalided) with tuberculosis, the intracutaneous

tuberculin test was applied at eleven general hospitals to over one thousand nurses, staff nurses and sisters who volunteered for the purpose. The tests were made with a single injection of "tuberculin P.P.D." in the way described by Anderson and Harvey.[8]

Their reactions to the test are set out in Tables I, II and III. In Table I the proportions of nurses infected in each quinquennial age group are shown, while in Figure I these proportions are compared with the proportions infected in corresponding age groups of young women following occupations other than nursing. It is shown that in the 15-19 year age group some 23% of the nurses are infected (not, of course, diseased) as against 29% of the young women in the population at large. (This may be because a large proportion of the younger nurses had always lived in the country prior to entering hospital: a study has been made to compare the invasion by tuberculosis of the urban and rural populations of New South Wales, and will be reported in a subsequent communication.) The rate of infection is more rapid in the nurses than in the other young women, and in the 25-29 year age group about three-quarters of the nurses are infected as against three-fifths of the others.

It may be argued that many of the "other young women" of Figure I are not subjects comparable with the nurses in that many have married in their twenties and become domesticated again, so that they could be expected to have less exposure to tuberculous contagion even than the general population.

TABLE I.

Reactions to the Intracutaneous Test of 1,025 Nurses in General Hospitals Grouped According to Age.

Ages.	Number Tested.	Number of Reactors.	Reactors *per centum*.
15 to 19	254	60	23·6
20 to 24	553	270	48·8
25 to 29	172	120	69·8
30 to 34	26	19	82·6
35	20	19	
Total	1,025		

TABLE II.

Reactions to the Intracutaneous Tuberculin Test of 1,025 Nurses in General Hospitals Grouped According to Length of Nursing Experience.

Years of Nursing Experience.	Number Tested.	Number of Reactors.	Reactors *per centum*.
0 to ½	361	103	31·3
½ to 1	123	48	39·0
1 to 1½	93	43	46·2
1½ to 2	99	61	61·6
2 to 2½	63	40	63·3
2½ to 3	58	30	51·7
3 to 3½	52	34	65·4
3½ to 4	64	46	71·9
4 and over	112	81	72·3
Total	1,025		

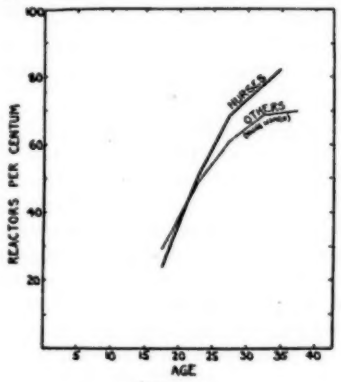

FIGURE I.

Reactions to the intracutaneous tuberculin test of 1,025 nurses (from Table I) and of a large number of young women in the population at large (from another paper by the author[9]) grouped according to age.

In Figure II where the proportions of infected nurses are compared with the proportions infected in the population at large, a closer correspondence of the two curves is seen. However, this does not alter the fact that nurses are being infected during their training on a substantial scale, and to a greater extent than might have been expected had they followed other occupations. In Table II the degree of tuberculous infection is correlated with the length of the 1,025 nurses' nursing experience, and in Table III, which is illustrated by Figure III, the reactors in each annual group of trainees are placed in age

TABLE III.

Reactions to the Intracutaneous Tuberculin Test of 1,025 Nurses in General Hospitals Grouped According to Age and Length of Nursing Experience.

Ages.	First Semester.			Second Semester.			Second Year.			Third Year.			Fourth Year.			Staff Nurses, Sisters.		
	Number Tested.	Number of Reactors.	Reactors *per centum*.	Number Tested.	Number of Reactors.	Reactors *per centum*.	Number Tested.	Number of Reactors.	Reactors *per centum*.	Number Tested.	Number of Reactors.	Reactors *per centum*.	Number Tested.	Number of Reactors.	Reactors *per centum*.	Number Tested.	Number of Reactors.	Reactors *per centum*.
15 to 19	194	41	21·1	47	16	34·0	10	2	—	1	0	—	1	0	—	1	1	—
20 to 24	135	46	34·1	62	25	40·3	152	79	52·0	96	55	57·3	80	52	65·0	28	13	46·4
25 to 29	31	16	51·6	14	7	50·0	26	20	76·9	22	15	68·2	28	24	85·7	51	38	74·5
30 to 34	2	0	—	0	0	—	3	2	—	1	0	—	6	5	—	14	12	85·7
35 and over	0	0	—	0	0	—	1	1	—	0	0	—	0	0	—	19	18	94·7
Totals	362	103		123	48		192	104		120	70		115	81		113	82	

groups also. It is seen that the senior nurses are much more heavily infected than the junior nurses, not only because their average age is greater, but age for age.

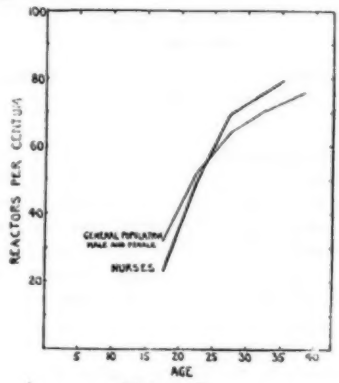

FIGURE II.

Reactions to the intracutaneous tuberculin test of 1,025 nurses (from Table I) and of a representative sample of the population at large (from another paper by the author[99]) grouped according to age.

That there is no significant difference in the degree of infection of nurses in the hospitals of Sydney, Newcastle and the country is shown by

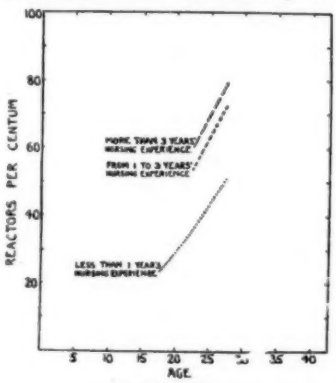

FIGURE III.

Reactions to the intracutaneous tuberculin test of 1,025 nurses in general hospitals grouped according to length of nursing experience (from Table III).

Table IV, which is illustrated by Figure IV. It would be of the greatest interest and value to have a comparative series of the reactions to the test of nurses working in children's hospitals. Unfortunately, the directors of a large children's hospital who were twice approached, could not be persuaded to allow tuberculin testing of their nurses even on a voluntary basis. It is here suggested that an intramural worker may be more successful in overcoming the scruples of authority, and it is hoped that statistics from a large children's hospital will be soon forthcoming.

Further Investigations Suggested.

It has been shown that among nurses in general hospitals in Sydney a larger proportion becomes infected by tuberculosis and this more rapidly than among other girls of the same age in other occupations. Heimbeck[1] has shown that under

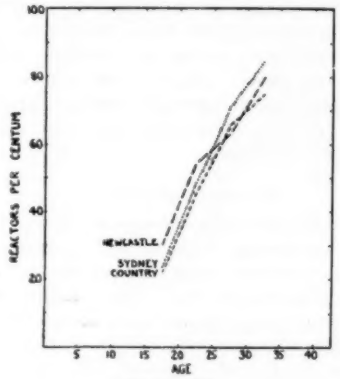

FIGURE IV.

Reactions to the intracutaneous tuberculin test of 1,025 nurses grouped according to the location of their hospitals (from Table IV).

Norwegian conditions this manner of tuberculous infection is relatively often followed soon by tuberculous disease of an extraordinarily severe and fatal type. His finding is that among those who commence to react to the cutaneous tuberculin test during their nursing training, the morbidity rate is 34%, as against 1% among those who reacted at the commencement. It is the author's impression that the morbidity from tuberculosis in our nurses is a small fraction of that which occurs in Heimbeck's nurses. Nevertheless it is necessary to replace impressions by facts. It is desirable to follow up the present work in the manner suggested in the section on prevention of disease, by further observation of the nurses who have been tested in this survey. It would be of

TABLE IV.

Reactions to the Intracutaneous Tuberculin Test of 1,025 Nurses in General Hospitals Grouped According to the Location of their Hospital.

Ages.	Hospitals about Sydney.			Country Hospitals.			Newcastle Hospital.		
	Number Tested.	Number of Reactors.	Reactors *per centum.*	Number Tested.	Number of Reactors.	Reactors *per centum.*	Number Tested.	Number of Reactors.	Reactors *per centum.*
15 to 19	213	50	23·5	28	6	21·4	13	4	30·8
20 to 24	418	203	48·6	73	33	45·2	62	34	54·8
25 to 29	136	97	71·3	17	11	65·3	19	12	63·2
30 and over	33	28	84·8	8	6	75·0	5	4	80·0
Totals	800			126			99		

value also if an appropriate body (such as perhaps the National Health and Medical Research Council) would obtain from the authorities of all large hospitals in Australia information concerning the incidence of tuberculosis among nurses in training. It is not likely that information would be withheld from an inquiry under the auspices of a body such as the council, if an assurance was given that the statistics of individual hospitals would not be published under the name of the hospitals. The council's present inquiry into the occupations of tuberculous patients might well be coordinated with this work.

It is finally desirable to systematize and unify this whole work in order that statistics may be prepared of the true incidence of tuberculous morbidity in nurses in this country, to have means by which it can be predicted and as far as possible minimized, and to have evidence which will enable anxious hospital authorities to decide whether they should adopt more active measures for the control of contagion and infection.

Acknowledgements.

It is a pleasure to acknowledge my great indebtedness to the following ladies and gentlemen, who helped me in various ways, especially by enabling me to get in touch with the large number of sisters and nurses who volunteered themselves as subjects for the Mantoux test: Dr. A. B. Lilley (General Superintendent, Royal Prince Alfred Hospital); Dr. K. W. Starr (Medical Superintendent, Newcastle Hospital); Miss P. M. Machin (Matron, Royal North Shore Hospital of Sydney); Miss M. Sirl (Sister Tutor, Royal North Shore Hospital of Sydney); Miss M. Alt (Home Sister, Royal Prince Alfred Hospital); Miss D. Armstrong (Matron, Eastern Suburbs Hospital, Sydney); Dr. K. B. Armstrong; R. B. Golsby, Esquire (Secretary, Bathurst District Hospital); Dr. A. S. Johnson (Deputy Superintendent, Royal Prince Alfred Hospital); Miss A. M. Kellett (Matron, Sydney Hospital); Miss E. Kerr (Matron, Orange Base Hospital); the Matrons, Bathurst District Hospital, Blue Mountains District Hospital, Maitland District Hospital, and the Western Suburbs Hospital, Sydney); Dr. Brooke Moore; Miss K. Siddins (Matron, Auburn District Hospital).

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FACTORS AFFECTING THE VITAMIN C (ASCORBIC ACID) CONTENT OF FRUIT AND VEGETABLES.[1]

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SEVERAL investigators have shown that the vitamin *C* (ascorbic acid) content of fruit and vegetables is considerably reduced by storage.

Biological experiments carried out on guinea-pigs by E. Batcheldor[1] showed that the vitamin *C* potency of "delicious" apples varied with the length and mode of storage. No loss of vitamin *C* was evident after six months' storage at 32° F. A loss of one-sixth occurred during the first three months of storage at 45° F., and a loss of one-quarter after six months' storage at this temperature.

That visible light plays a large part in the destruction of the vitamin *C* content of milk was demonstrated by Kon and Watson[3], who found that after the milk had been exposed to sunshine for two hours, the reduced form of ascorbic acid was nil, whilst the dehydroascorbic acid was about one-third of the total original value.

To test these observations under local conditions a series of determinations was carried out on the ascorbic acid content of Queensland fruit and vegetables, different methods of storage being used.

Method.

The vitamin *C* content was determined by the chemical method of Birch, Harris and Ray,[2] as modified by Leggett and Robinson.[4] This method consists in extracting the vitamin from the material with N/10 trichloracetic acid and sand, centrifuging, removing an aliquot, adjusting its pH to 2·5 and titrating with the dye (2:6 dichlorphenol-indophenol). Estimations were made of the reduced form and of dehydroascorbic acid.

Effect of Storage.

Samples of fruit and vegetables were stored in the following ways: (*a*) on the shelf, exposed to air at room temperature (approximately 25° C.); (*b*) in the refrigerator, exposed to air; and (*c*) (in the case of oranges) wrapped in paper and kept in sealed tins at room temperature.

The water loss was determined and a correction was made for it in every case. The results are shown in Tables I and II.

Cabbage maintained its vitamin *C* content over a period of five days in both methods of storage. The rising figure may have been due to faulty correction for water loss or more probably to liberation of bound vitamin *C*, as in cooking. The biggest difference is apparent in peas, which lost 50% in four days at room temperature, but only 14% when

[1] This work was recently carried out by the authors for the Queensland Nutrition Council.

TABLE I.

Effect of Storage on Vitamin C Content.

Substance.	Number of Samples.	Method of Storage.	Time of Storage.										Final % Loss of Vitamin C.	Appearance.
			0 day. Mean.	1 day.		2 days.		3 days.		4 days.		5 days.		
				Mean.[1]	Corrected Mean.[1]	Mean.	Corrected Mean.	Mean.	Corrected Mean.	Mean.	Corrected Mean.	Mean.	Corrected Mean.	
Peas ..	4	On shelf exposed to air.	28·6	25·9	23·6	—	—	23·9	16·1	22·9	14·1	—	—	51	Very withered at end of three days.
	4	In refrigerator exposed to air.	28·6	26·7	25·4	—	—	28·8	25·7	28·5	24·6	—	—	14	Green, but had lost part of their crispness.
Beans	4	On shelf exposed to air.	30·0	31·6	28·2	—	—	27·0	20·0	30·2	20·3	—	—	32	Similar to peas.
	4	In refrigerator exposed to air.	30·0	34·8	33·9	—	—	29·3	26·4	29·3	25·6	—	—	15	
Cabbage	4	On shelf exposed to air.	47·7	53·8	51·5	60·3	56·4	58·7	53·8	64·4	57·4	63·1	54·9	—	Leaves yellow and limp.
	4	In refrigerator exposed to air.	47·7	56·9	56·0	54·3	52·9	55·0	52·7	57·5	54·4	53·6	50·0	—	Head green, but some loss of firmness.
Pawpaw[2]	4	On shelf exposed to air.	73·0	73·0	71·8	75·0	72·8	67·9	65·0	63·2	59·3	—	—	19	Soft.
	4	In refrigerator exposed to air.	73·0	70·3	69·9	70·5	69·8	62·8	61·9	56·6	55·5	—	—	24	Firm.

[1] The amounts in these columns are expressed in milligrammes per 100 grammes.

[2] The pawpaws were fully ripe at the time of testing. In each case daily samples were taken from the original fruit or vegetable. The corrected mean represents the ascorbic acid content after allowance has been made for water loss.

TABLE II.

Effect of Storage on the Vitamin C Content of Oranges.[1]

Method of Storage.	Number of Samples.	Time of Storage.												Final % Loss of Vitamin C.	Appearance.
		0 day. Mean.[2]	3 days.		7 days.		10 days.		14 days.		17 days.		21 days.		
			Mean.[2]	Corrected Mean.	Mean.	Corrected Mean.	Mean.	Corrected Mean.	Mean.	Corrected Mean.	Mean.	Corrected Mean.	Mean.	Corrected Mean.	
On shelf exposed to air.	4	56·1	57·6	54·2	48·9	42·4	50·2	40·6	53·7	39·5	48·3	30·5	45·1	22·7	60	Drying and shrinkage of skin, with general hardening.
In refrigerator exposed to air.	4	56·1	58·0	56·9	54·3	52·5	54·8	52·2	55·8	51·8	56·3	51·3	58·5	52·3	7	Maintained original appearance.
Wrapped in paper in sealed tins.	4	56·1	56·6	55·4	57·3	56·3	57·9	56·4	57·1	55·1	57·2	54·2	56·4	52·8	6	Maintained original appearance.

[1] All the oranges were picked from the same tree. The corrected mean represents the ascorbic acid content after allowance has been made for water loss.

[2] The amounts in these columns are expressed in milligrammes per 100 grammes.

stored in the refrigerator for the same period. With beans also refrigeration proves to be the better method of storage. In the case of pawpaw the small difference in vitamin *C* loss with the two methods may be attributed to sampling.

Oranges stored for three weeks practically maintained their vitamin *C* content when stored either (*a*) in the refrigerator or (*b*) wrapped in paper in sealed tins at room temperature. This was in great contrast to the very considerable loss of approximately 60% which occurred when they were kept exposed to air at room temperature. The method of storing oranges by wrapping them in paper and keeping in sealed tins, thus reducing vitamin destruction, should prove valuable in certain parts of Queensland when this fruit is kept for some time after being picked and refrigerators are not available.

Effect of Sampling on Vitamin C Content.

That the vitamin is not distributed evenly throughout the plant is demonstrated by estimations made on different portions of the same cabbage and pawpaw. Hence sampling plays a large part in vitamin *C* determinations. Equally important is it that the valuable outer parts of these articles should not be discarded. The results are shown in Table III.

In the case of pawpaw, the outer portion just under the skin is the richest source of vitamin *C*; the content gradually decreases as one passes to the innermost portion.

The outer green leaves of cabbage have a considerably greater antiscorbutic value than the inner leaves. The vitamin *C* content appears to be correlated with the amount of exposure to sunlight.

TABLE III.

Foodstuff.	Outer Portion (just under Skin) or Leaves. (Total ascorbic acid; milligrammes per 100 grammes.)	Middle Portion or Leaves. (Total ascorbic acid; milligrammes per 100 grammes.)	Inner Portion or Leaves. (Total ascorbic acid; milligrammes per 100 grammes.)
Pawpaw 1..	118	—	100
2..	159	111	94
3..	178	105	94
4	126	106	106
5..	112	100	98
Cabbage 1..	130	47	51
2..	140	48	54
3..	150	48	61
4..	131	55	68
5..	91	64	64

Effect of the Stage of Ripeness on the Vitamin C Content of the Pawpaw.

Determinations were carried out on green, half-ripe and ripe pawpaws picked from the same tree. The results in Table IV clearly demonstrate that the ascorbic acid content is directly proportional to the stage of ripeness.

TABLE IV.

Effect of Degree of Ripeness on Vitamin C Content of Pawpaw.

Foodstuff.	State of Ripeness.		
	Green. (Milligrammes per 100 grammes.)	Half Ripe. (Milligrammes per 100 grammes.)	Ripe. (Milligrammes per 100 grammes.)
Pawpaw—			
1 ..	31	82	107
2 ..	48	59	106
3 ..	38	68	118
4 ..	30	50	93

Summary.

Tests carried out on the loss of vitamin *C* when different methods of storage are used indicate that in the case of peas, beans, pawpaws and oranges, the destruction with refrigeration is considerably reduced in comparison with storage at room temperature (approximately 25° C.) and with exposure to air. In the case of oranges, however, if the fruit is wrapped in paper and kept in air-tight containers at room temperature, the loss of vitamin *C* is considerably reduced, being practically equivalent to that occurring during storage in a refrigerator.

Figures are given showing the effect of sampling on the vitamin *C* content of pawpaws and cabbage. They indicate an increase in value from the inner to the outer portions.

The vitamin *C* content of pawpaw depends upon the stage of ripeness, being highest in the fully ripe fruit.

Acknowledgements.

We wish to thank Professor D. H. K. Lee and Mr. H. J. G. Hines, of the Department of Physiology, University of Queensland, for making available the facilities of the department for this investigation.

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A TECHNIQUE FOR THE ESTIMATION OF GAS TENSIONS IN THE TISSUES BY THE GAS DEPOT METHOD.[1]

By ERIC W. SIBREE.

(From the Department of Medicine, University of Sydney.[1])

IN a previous article[(11)] a general discussion of gas tensions in the tissues was given and the present article describes details of the technique used in the present investigation; it is a modification of the gas depot method employed extensively by J. Argyll Campbell[(3)] in animals. In most animals estimations of gas tensions can be made with ease, so that details of technique are relatively unimportant; but in man estimations are comparatively difficult, so that technique must be studied carefully, the essential difficulty being the handling of small volumes of gas.

A few investigators have made studies in human subjects using modifications of Campbell's technique; but these studies are fragmentary. This is doubtless due not only to essential limitations of the gas depot method in human tissues, but also to the difficulty of technique in such subjects. The technique here described in detail has been found satisfactory for both animal and human investigations, and involves the collection, transference and analysis of small samples of gas, if necessary without loss of volume. The rabbit has been used satisfactorily for animal studies, which are of value for comparison with the results obtained in man.

The method will be considered under the successive phases of operation.

The Formation of Gas Depots in the Tissues.

The formation of gas depots in the tissues is the simplest step in the gas depot method, and consists essentially in the introduction of gas under the skin or into other tissues under investigation. Room air and commercial nitrogen are the gases most readily available, and they are quite satisfactory. For routine work air is used for rabbits and nitrogen for the human subject.

Rabbits.

The air injected into rabbits is introduced directly, by means of an ordinary Higginson's syringe, without being filtered, warmed or moistened (Figure I). The outlet tube of the syringe is fitted with a wide-bore hypodermic

[1] Work done with the aid of a grant from the Australian National Health and Medical Research Council.

needle; if necessary, a metal or glass connexion may be interposed to give an airtight joint. When it is desired to inject gas other than air, the gas is introduced into a rubber bag, such as a football bladder, the outlet of which is connected to the inlet tube of the Higginson's syringe.

For initial injections from half to one litre is used under the skin, or about half those volumes in the peritoneal cavity, these sites being the best for gas tension studies. The approximate volumes are judged from the volume of the syringe. No special precautions are necessary in the introduction. It is convenient to keep the animal's fur clipped at the sites of the gas depots; but this is not essential, nor is sterilization of the skin necessary before the injections or withdrawals. No cases of infection have been noted.

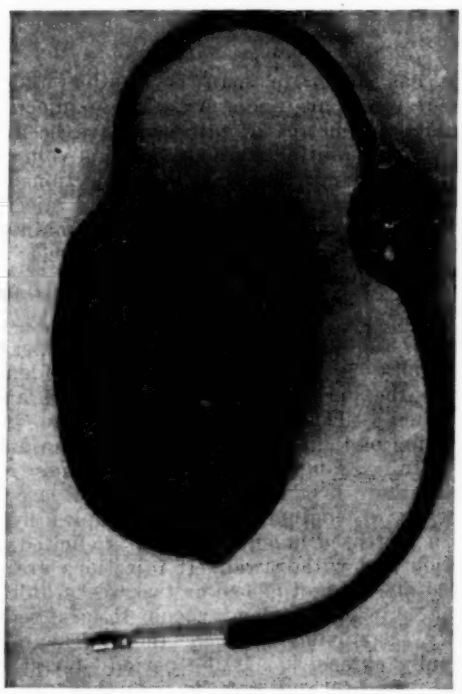

FIGURE I.

Apparatus for the injection of gas into the tissues of rabbits, showing Higginson's syringe with glass connexion and hypodermic needle and rubber bag to contain the gas for injection.

When a large volume of gas is injected anywhere under the skin of a rabbit the gas will collect mostly on the back, especially towards the shoulders; so that no special site for injection is necessary, but the centre of the back is most readily available. For intraperitoneal injections the centre of the abdomen is the most convenient site. Occasionally an abdominal injection may be made accidentally into the intestine. This is best avoided by commencing injection as the needle is piercing the abdominal wall. Generally a characteristic bubbling sound is made as the gas enters the peritoneal cavity. Injections should be made slowly to avoid causing undue tension. The tension produced is the best guide to the volume to be injected. The gas depot should feel "baggy" at the end of injection; this assures sufficient volume for withdrawals without causing sufficient pressure to interfere with circulation.

It is unnecessary and undesirable to restrain the animal in a box or by other mechanical means during injections and withdrawals. With experience the animal may be held quietly and all manipulations can be made by one operator.

Human Subject.

In man the subcutaneous tissues are the only site used for gas depot formation. Unlike the very loose skin of the rabbit, under which a large gas depot is readily formed, the human skin is comparatively closely bound down to the underlying structures. For this reason only small volumes, up to about fifty cubic centimetres of gas, can be injected regularly without causing considerable discomfort from tension. Further modifying factors are the greater necessity for asepsis and the psychological effect, particularly in hospital patients, of unnecessary apparatus.

Commercial nitrogen has been used satisfactorily for routine injections in the human subject. I have repeatedly injected unfiltered room air into my own subcutaneous tissues without infection; but it is an undesirable procedure for general work, owing to this possibility.

The nitrogen is first bubbled through water and is then stored over boiled water in a glass jar (Figure II). Such a container is conveniently provided with three outlets, one for withdrawal of samples through a rubber stopper, the second for a mercury manometer, and the third connecting with a water reservoir which fills the gas jar as gas is removed, maintaining a positive pressure. When nitrogen was stored in a rubber bag it was found

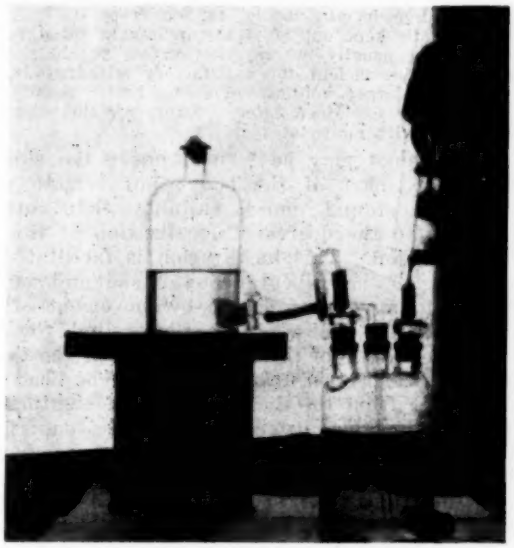

FIGURE II.

Gas jar used as reservoir for nitrogen for injection into human subcutaneous tissues, showing mercury manometer, rubber cap with hypodermic needle from which gas is being withdrawn into the "Record" syringe, and water reservoir (in the background) which automatically fills the gas jar as gas is removed.

that the gas apparently became contaminated by the rubber, and after repeated injections over a period of several months this gave rise to sensitization phenomena. I noticed such phenomena in myself, but only after constant injections over a long period. The symptoms did not appear when the rubber bag reservoir was discarded and storage over water substituted. No such phenomena have ever been elicited in subjects used for short periods, even by gas from the rubber bag.

For simplicity of operation and to prevent undue apprehension on the part of the subject, an ordinary twenty

cubic centimetre "Record" syringe is used for injections. Gas from the gas storage jar is withdrawn through a rubber cap by means of a wide-bore needle. The gas is injected directly from the syringe through a fine hypodermic needle into the subcutaneous tissues.

The only precautions to be adopted are those for any hypodermic injection. The syringe and hypodermic needle are first cleaned with methylated spirit and distilled water. The skin is cleaned also with methylated spirit and is allowed to dry before the introduction of the needle. The needle is introduced through the skin until the point is felt to be freely movable in the connective tissue between the tough superficial layers and the deep fascia. Any obvious superficial blood vessels are, of course, avoided. The syringe is then fitted to the needle and the plunger is withdrawn slightly to make negative pressure in the syringe, so as to be certain that a blood vessel has not been entered. A fine-bore hypodermic needle serves a double purpose in assuring minimum discomfort and in making the injection necessarily slow.

As gas is injected it will be seen swelling under the skin and tracking away in various directions, often along the course of superficial veins. The accompanying sensations in the subject vary from a consciousness of slight swelling locally and a crawling sensation to occasional sharp stinging pain, often at some distance from the needle. Generally there is no discomfort; sometimes the injection is not felt. A few hours after an initial injection tenderness develops over the depot, lasting from one to two days, and thereafter subsiding, although slight discomfort to the touch may remain. In the forearm an initial injection may give rise to considerable aching during this reaction period. When more than 20 cubic centimetres are to be injected, the syringe is disconnected from the hypodermic needle, refilled from the gas jar, which can be kept out of sight, and again fitted to the needle. But usually, when injections of 20 cubic centimetres are insufficient for satisfactory withdrawals, the injection of larger volumes gives no better results, for reasons to be explained later. After injection the part is swabbed with methylated spirit.

A gas depot may be formed under the skin of almost any part of the body; but certain sites have been found more suitable than others, according to the degree of localization of the gas after injection. This localization is facilitated by looseness of the subcutaneous tissue and is not affected to any great degree by movement of the part, as in the limbs. In the lower limb the best site is the middle of the medial aspect of the thigh; the lateral aspect is unsatisfactory. The head and neck suffer from the disadvantages of tightness of the skin or the proximity of large superficial blood vessels and the formation of temporary disfigurement.

On the trunk the best depots have been obtained on the anterior surface of the abdomen, particularly in the lower half, where the skin is moderately loose. Routine work has been carried out on depots formed in the left lower quadrant of the abdomen, a little below and medial to the iliac spine. This site possesses the advantage that the skin in this area has a temperature close to that of the general body temperature.

The forearm also has been used. The best area is the dorsal aspect of the upper third. The anterior surface does not form a depot readily. The forearm is a convenient site for certain types of experiment, such as changes in local temperature and local circulation, and has been used almost exclusively for such work. The abdominal sites have been used mostly when general bodily changes were involved.

Maintenance of the Gas Depot.

Rabbits.

Owing to the large volumes of gas which can be injected and the ease of obtaining samples, maintenance of the gas depots is readily accomplished in rabbits. Occasional samples can be obtained for several days after an injection; but when repeated samples are required daily, gas must be added every day or second day, 200 to 300 cubic centimetres for skin or abdomen generally being required. Gas will be palpable for a week or longer under the skin when injections are discontinued; absorption of gas from the peritoneal cavity is more rapid.

Human Subject.

The gas depot in man is smaller and more frequent refills are necessary. Daily injections of 20 cubic centimetres of nitrogen have been found effective as a routine method; but this volume may be varied for individuals. These injections should be made into the area of the previous injection to avoid opening up fresh tissues. All maintenance injections are made in the same manner as the initial injection.

Periods for which a Gas Depot may be Used.

In the rabbit the subcutaneous and peritoneal depots may be maintained and used indefinitely. In man the period of continuous use of any site is limited. This limit is set by the gradual thickening of the site, which increases the difficulty of obtaining samples until this becomes impracticable; there is also a concurrent increase in the frequency of contamination of samples by blood and an increased amount of pain is elicited during injection and withdrawal. If injections are recommenced after a rest of one or two weeks, thickening of the skin and subcutaneous tissues reappears rapidly, particularly when the thickening was originally extensive. If the period of rest is prolonged to several weeks, further injections frequently tend to remain at first more localized, owing to the deposition of fibrous tissue. Sometimes the fibrous tissue at the site of a previous depot is sufficiently dense to make injection into the area very difficult. When a second series of injections in any subject is required it is best to use a different site for the new depot.

Seevers and his co-workers,[8][9] using the anterior abdominal wall, maintained depots for as long as six weeks; but he does not refer to the condition of the subcutaneous tissues at the end of such a period, nor to any additional difficulties in sampling during the later stages of observation.

Withdrawal of Samples.

Withdrawal is the most difficult step in the gas depot method. The requirements for satisfactory sampling are the following: (i) The sample as analysed must represent the composition of the gas depot, requiring freedom from contamination by air, blood and serum. (ii) As large a volume as necessary for accurate analysis should be with-

drawn. (iii) Ease and rapidity of withdrawal, with simplicity and portability of apparatus for clinical work, are essential. (iv) Ease of transference of the sample from the collecting apparatus to the gas analyser, without contamination by air or appreciable loss of volume, is required.

In rabbits, owing to the large volume of the gas depots and their "baggy" nature, these requirements are readily satisfied. Repeated sampling can be performed, 10 cubic centimetres or more being obtained regularly; this is sufficient for introduction into and analysis in a Haldane gas analyser. Gas may be wasted, if necessary, in washing out dead space in the collection or transference of samples.

In man, however, as the depot is small in volume, and often only a thin layer of gas is dispersed through the subcutaneous tissues, there are considerable difficulties. Samples of only about 0·5 cubic centimetre can generally be obtained in routine work, though occasionally one may obtain samples several cubic centimetres in volume.

The samples obtainable are not only smaller, but are more difficult to withdraw than are those from a rabbit. A microanalyser is required, the utmost economy must be observed to prevent loss of volume in collection and transference of samples, and great care is necessary to prevent contamination by air.

Three forms of apparatus have been tried: (i) a glass vessel filled with fluid, into which samples are drawn by withdrawal of fluid; (ii) a syringe filled with glycerol or paraffin; (iii) a modified tonometer.

A glass collecting vessel fitted with rubber connecting tubing may be used for animals, as in experiments by Campbell[3] and Schott;[7] but it is unsuitable for human investigations, owing to dead space between the glass container and the hypodermic needle, which must be washed out. The apparatus is also cumbersome and not easily handled, even with animals. This method was used for human subjects by Bazett and Sribyatta,[2] but their observations were mostly in gas depots an hour or two after the injection of gas. The large volumes which they obtained on sampling were sufficient to enable washing out of dead space; but such volumes cannot be expected in routine work. The same objection applies to the collecting apparatus[10] used by Seevers for human subjects; this consisted of glass bulbs filled with mercury, connected to a hypodermic needle by rubber tubing.

A syringe may be used in human investigations as in the observations of Ellman and Taylor;[5] but this method has great disadvantages, in that it is very difficult to be certain that no air leaks around the plunger of the syringe, since considerable negative pressure must be maintained during sampling. It is also difficult to manipulate a syringe while maintaining sufficient pull on the plunger. Great care must be taken also to prevent air from being admitted through the needle after withdrawal from the skin, since there is no tap with which to lock the sample. F. Meyer[6] used a syringe with a needle permanently connected, filling the sampler with acidified glycerol. His figure for normal oxygen tension in the tissues (37 millimetres of mercury) is, however, high as compared with findings by Seevers[8] of an average oxygen tension of 22 millimetres of mercury. It is possible that contamination by air in this method of withdrawal was partly the cause. Campbell[4] also used a syringe for sampling gas depots of small volume in the lateral ventricles of the brain of rabbits. In this type of experiment the syringe method may be more applicable, owing to greater ease of withdrawal of samples.

A Modified Tonometer.

I have used a modified tonometer in which all dead space can be eliminated during the collection and transference of samples. This method also suffers from disadvantages in human investigations, but it has been found more satisfactory than other methods tried.

A glass tonometer (Figures III and IV) of a capacity of about 15 cubic centimetres with two-way capillary taps is used, constructed to the same pattern as the usual large tonometers used for gas sampling. One outlet tube ends

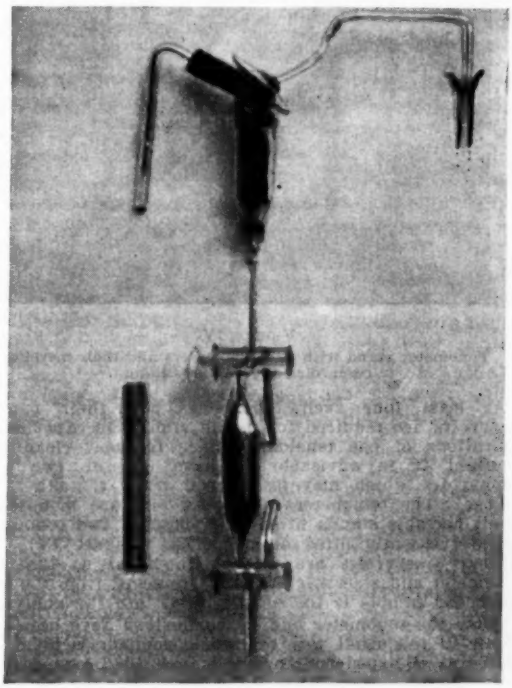

FIGURE III.

Modified tonometer used for withdrawal of gas samples from gas depots with hypodermic needle attached to the ground glass joint, in position for the ejection of a gas sample into the introducer (above). The introducer is assembled for use. The cup of the inlet tube of the analysing burette is shown diagrammatically. The rule beside the tonometer is graduated in centimetres.

in a ground glass joint to take a Luer fitting needle. The other end of the tonometer is connected by rubber pressure tubing to a mercury reservoir. The capillary tubing of the taps should be free of any sudden constriction or enlargement of its bore at the needle joint or at the tap, since gas pockets are apt to form where the bore is uneven.

The bore should be about one millimetre. The capillary tubing with the needle connexion should be about two inches in length and the body of the tonometer about three inches in length to permit of easy handling.

The hypodermic needles used are 23 gauge. This bore is the smallest that will allow sampling without frequent blocking of the needle in the tissues. Needles of larger bore and needles with side holes have been tried, but they give no better results, cause more pain and must cause greater tissue damage.

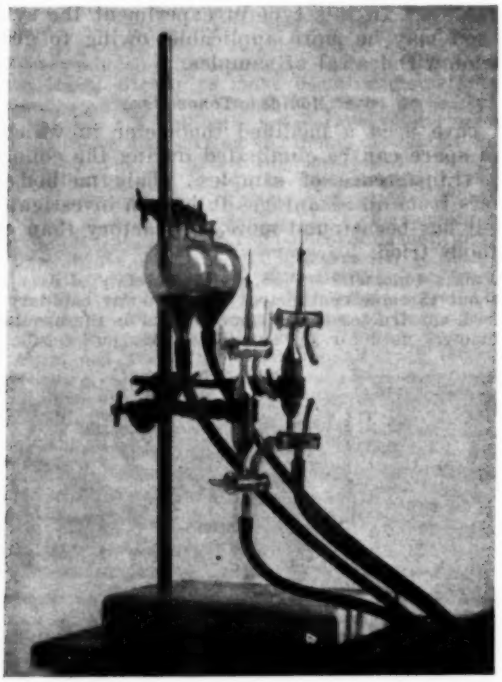

FIGURE IV.
Tonometer stand with two tonometers and their mercury reservoirs and rubber tubing.

At least four such tonometers and their mercury reservoirs are required for use in studies in experimental alterations of gas tensions. Since frequent cleaning is required it is advisable to have one or two extra tonometers which may be cleaned while the others are in use. The tonometers and reservoirs are mounted on small portable stands fitted with clamps; two tonometers are conveniently fitted on one stand (Figure IV). The mercury reservoirs are fitted with hooks so that both tonometer and reservoir are easily removed from the stand when the sample is to be introduced into the analyser.

After the tonometer and its connexions have been freed of air in the usual way the whole sampler is filled with mercury. A hypodermic needle is now filled carefully with glycerol and is then fitted to the tonometer, the joint being smeared with "Vaseline". The glycerol is now forced out of the needle by running mercury through it. If the glycerol is not used a very small bubble of air will generally remain in the needle even when mercury is run through it. Immediately before use the tonometer is evacuated, except for about half a cubic centimetre of mercury which remains in the tonometer, and the tap to the reservoir is then closed. The needle is wiped with a swab containing methylated spirit, the skin is cleaned in the same way and the needle is inserted into the gas depot (Figure V). The tap at the needle end of the tonometer is opened, and this connects the needle to the evacuated tonometer. In the rabbit, the tonometer is filled in a few seconds; but in the human subject usually the needle has to be moved about from point to point for some time to collect bubbles of gas. In the position in which the tonometer is held the small amount of mercury left in the tonometer will lie over the entrance from the tap connected with the needle, so that the flow of gas from the depot to the tonometer may be seen bubbling through the mercury. With experience, one may judge the approximate volume of the sample obtained by this flow through the mercury. At the end of collection, the tap at the needle end of the tonometer is closed, the needle is withdrawn from the skin and the part is cleaned with spirits. The needle is then removed from the tonometer. During sampling, the mercury reservoir attached to the tonometer should be kept at a level lower than the tonometer. Should the tap connecting to the reservoir be opened accidentally during sampling there will still be a negative pressure in the tonometer preventing mercury from being injected into the tissues.

The only volume of gas which is lost is that lying in the needle and the short connecting capillary tube leading to the tonometer. In the rabbit, when more than 10 cubic centimetres of gas have been obtained, this loss is of no moment, and in man, where the gas sample is still under greatly reduced pressure, the volume in the needle and connecting tube is insignificant.

FIGURE V.
Withdrawal of sample from an abdominal depot. The mercury reservoir is at a lower level on the floor.

When the sample is taken from a rabbit, the tap at the reservoir end of the tonometer is then opened through the side arm, and any small amount of air, which sometimes collects during evacuation of the tonometer, is run out. This tap is then opened to the tonometer, so that a slightly positive pressure is induced in the gas sample, and is maintained thus until the sample is to be transferred to the analysing apparatus. After a sample from the human subject has been collected much greater care is required to ensure that no small bubble of air is carried into the tonometer during the admission of mercury. Even after any visible bubble of gas below the tap leading to the reservoir has been washed out, as described above, other

small bubbles, probably of air, may be carried up from the rubber tubing into the tonometer by the rush of mercury when the tap is opened to the tonometer. To avoid this possibility, the tonometer inlet tube with the ground glass joint is now filled with mercury sucked up from a beaker into the side arm. In this way, under direct observation, all air may be expelled from this tube. When the tonometer inlet has thus been made gas free the tap is turned to connect the tonometer with the inlet which is still in the mercury. Mercury now flows up into the tonometer and is allowed to rise as high as it will, and the inlet tap is then closed. The tubing immediately below the tonometer is next evacuated, the tonometer tap being closed, and any gas brought up is expelled through the side arm. This is repeated until no more gas is carried up. The tap connected with the mercury reservoir is opened until a slight positive pressure is induced. The sample is now ready for introduction into the analyser.

Precautions.

Scrupulous care must be taken throughout to prevent contamination by air, either by the leaving of air in the tonometer and its connexions before collection of the sample or by admission of air after collection. The tonometer must be kept clean and the taps well greased. Grease must be prevented as far as possible from passing up into the capillary tubing, since gas pockets may form on it. The bore of the taps must be inspected before the final evacuation of the tonometer, to make sure that no small pockets of gas remain in them. It is in and close to the taps and within the needle that gas is most likely to remain in small bubbles. Since a sample may be only 0·5 cubic centimetre or less in volume, with carbon dioxide and oxygen percentage of about 7 and 3 respectively, a great error would result from contamination by even a minute volume of air having corresponding percentages of 0·04 and 20·93.

Another source of contamination of the samples is from blood or serous fluid withdrawn during samplings. The tendency to contamination in this way varies considerably, being seldom found in routine twenty-four hour samples from gas depots used for not more than two weeks. The tendency increases greatly after this period and with frequent sampling, often rendering a subcutaneous site unsuitable for further use. Ideally, no sample should be accepted having any trace of blood or serum, which under the reduced pressure in the tonometer will give off most of the contained gases. However, when the pressure is increased back to normal, gases will be reabsorbed, although not necessarily to the same extent. In practice, it has been found that very small amounts of body fluid withdrawn with a large sample do not alter appreciably the tensions of carbon dioxide and oxygen in the sample, as compared with a dry specimen. However, samples smaller than 0·5 cubic centimetre when thus contaminated, or large samples with large amounts of fluid, must be discarded.

From all glassware used in the collection and introduction of samples all traces of acid (as those found in cleaning mixtures) or alkali must be carefully washed out with plain water before use, in order to prevent permanent liberation or absorption of gases from body fluids.

Instead of running out the glycerol from the needle after it has been fitted to the tonometer, the glycerol may be left in the needle; but this tends to soil the tonometer and eventually the analysing apparatus, where it may cause changes in water vapour pressure. No ill effects have been noted from the presence of mercury in the needle as it is introduced through the skin. The negative pressure of the tonometer prevents mercury from flowing into the tissues during sampling. It is of interest to note that I have had a small amount of mercury under the skin at the site of one gas depot (accidentally injected in an early experiment) for many months without its having given rise to any symptoms. Mercury soon destroys the soldered joint found in some hypodermic needles. When this occurs the needle must be discarded.

Comparison of Methods of Sampling Gas Depots.

Sufficient has been stated of this and other methods of sampling to indicate the advantages and disadvantages of each method. In rabbits, the large volumes obtainable allow accuracy by many modified methods, but the evacuated tonometer technique here described has clear advantages in rapidity, simplicity and ease of handling. In the human subject, however, in whom samples are small and difficult to obtain, any method involving loss of gas by dead space must be ruled out. The choice lies between the use of a syringe and a modified tonometer in which all dead space may be filled with liquid. In the syringe method the lack of taps is both an advantage (in freedom from gas pockets) and a disadvantage, in that gas cannot be locked in the syringe after sampling. The tonometer can be thoroughly cleared of gas by evacuation before use, and samples may be left in the tonometer after collection for some time without fear of contamination or loss. The tonometer is light and easily handled and collects larger samples than are possible by the syringe method. The results obtained by the tonometer method justify the great care necessary in its use.

The Difficulty of Obtaining Samples from the Human Subject.

The greatest disadvantage of the gas depot method in the human subject is the difficulty of obtaining regularly samples sufficiently large for accuracy. This difficulty is variable from individual to individual and from time to time in the same gas depot. Several subjects, both normal and abnormal, have had to be discarded after as many as six unsuccessful attempts at daily withdrawal from a depot into which 30 cubic centimetres of gas had been injected daily. Frequently, from a depot which has been in existence for several days, it has been found impossible to withdraw a satisfactory sample after repeated attempts only an hour after the injection of as much as 100 cubic centimetres of gas. The explanation of this remarkable difficulty lies in the nature of the depot. Except in subjects with very little subcutaneous tissue, or for a short while after an initial injection,

the gas depot in the human subject consists usually not of a single discrete pocket of gas, but of a loculated, finely divided layer spread through the tissues. An idea of this state is obtained by pressing the chest piece of a stethoscope over the depot, when a multitude of fine crepitations will be heard. Here and there larger bubbles will be encountered during withdrawal, as seen by sudden rushes of gas into the tonometer.

The important practical result of the factors mentioned is that regular samples cannot always be obtained when desired, even for single daily estimations. This irregularity cannot be predicted with any certainty; of two individuals with apparently the same type of subcutaneous tissues, one may give good samples and the other none at all. In general, the greater the amount of adipose tissue, the harder it is to get samples. When experimental alterations of gas tensions in the human subcutaneous tissues are to be studied the difficulties are further increased. For such observations at least three consecutive samples are required; the first to give the normal tensions at that time, the second to note the primary experimental alterations and the third to note the reaction. Often as many as six samples are desirable. It will be obvious that it is impossible to conduct a long series of experiments on different subjects owing to the irregularity of obtaining samples. Also there is the mental factor; only those who are actively concerned in the research will have sufficient enthusiasm to stand the sometimes painful effects of sampling at more than infrequent intervals. The great bulk of the work in physiological and experimental alterations of gas tensions in the normal human tissues must be conducted on the experimenters themselves. For this reason, it would be desirable to have a group of two or three collaborators who should be thin-skinned literally but not figuratively. I have used my own subcutaneous tissues for most of the physiological changes to be published later. When possible other subjects have also been used. It is of great help that most of the human studies can be checked in animals such as the rabbit, in which, owing to the ease of sampling, experiments may be conducted with greater regularity, accuracy and control.

Transference of Gas Samples to Analysing Apparatus.

Samples from Rabbits.

For analysis of gas samples obtained from rabbits (12 to 15 cubic centimetres in volume) the usual Haldane gas analyser has been used. Introduction of the sample into the analysing burette is accomplished directly from the tonometer by a combination of the usual methods of introduction without loss of volume, and rinsing.

The needle connexion inlet of the tonometer is connected with the burette inlet by a simple glass capillary tubing bridge with two rubber joints; no tap is used on the connecting bridge. After an analysis most of the residual nitrogen is expelled from the burette through the connecting bridge, and several cubic centimetres of acidified water are drawn into the burette. The tonometer is then connected to the burette and some of the water from the burette is carried over and out through the tonometer side arm. One rinsing with one or two cubic centimetres of gas from the tonometer is desirable to wash out any bubbles of gas remaining in the rubber connexions and burette tap. The gas used for rinsing is then expelled down the tonometer side arm, followed by all the water, except the small volume that fills the capillary bridge. Gas from the tonometer is then admitted to the burette, and brings over with it the water filling the bridge. If this small volume of water is not left in the burette during analysis considerable difficulty may be experienced in obtaining an end point after absorption of oxygen. This is due to slow apparent increase in gas volume caused by the differences in water vapour pressure between the burette and the pyrogallol chamber.

It will be noticed that the tonometer is not disconnected from its mercury reservoir. The use of acidified water for filling the connecting bridge lessens the contamination of mercury by grease in the tap of the burette.

To check the accuracy of this technique two series of samples were taken from a gas reservoir in a flask through a rubber stopper. The exact routine of sampling, introduction and analysis was observed as with animal experiments. No extra care was taken beyond that observed in routine work. Two different gas mixtures were used, the composition in each case being within the range found in animal gas depots. From the first mixture ten consecutive samples were withdrawn and seven from the second. In each case, the greatest variation from the average found was 0·04%, corresponding to 0·3 millimetre of mercury partial pressure. The average composition was the same as that found on analysis of large samples taken from the gas reservoir in the usual type of gas sampling tonometer. The error found is very little greater than that of the Haldane analysis itself, and represents the total error of the modified tonometer technique.

When necessary, gas tensions in the depots in rabbits may be given to 0·1 millimetre of mercury, and changes of more than 0·3 millimetre of mercury may be regarded as having origin in the depots.

Samples from Human Tissues.

The small size of the gas samples from human subjects necessitates some form of microanalyser. I have used an instrument similar to that described by Bazett,[1] which has proved satisfactory. In this instrument the inlet for introduction of samples is a vertical capillary tube. Bazett used a rubber connexion during introduction, but the samples he used were considerably larger than those obtainable in routine work. Routine samples are too small to allow rinsing, and any rubber connexion must be discarded, owing to the impossibility of being certain that no small gas bubbles are caught in the tubing.

The following form of introducer has been found satisfactory for the introduction of samples of any size within the range of the analysing apparatus, without the use of rubber connexions.

The end of the inlet tube of the burette is enlarged to form a small cup, into the bottom of which the end of a connecting capillary tube is ground to form a loose ground-glass joint. This joint, protected by a little

"Vaseline" and a mercury seal, is air-tight and can be examined directly for gas pocketing.

The introduction apparatus can be understood best by reference to Figures III and VI. Figure VI shows the various parts of the introducer to which letter references are given below. These parts are shown assembled in Figure III, in relation to the tonometer and the inlet tube of the analysing burette (sketched in), as during the actual introduction of a gas sample. The apparatus consists of two parts. The connecting tube (CT) has a tap (T) which allows the connexion of the analysing burette, the inverted collecting cup (CC) and the tap side arm (TSA), in pairs. The bore of the tap is ⋀-shaped so that there is no possibility of dead space in which gas may pocket as in the ordinary T-bore tap. The inverted collecting cup fits loosely into and rests in the larger outer cup (OC) which is open at the top and constricted below to take a small rubber stopper (RS). This outer cup is held in a clamp attached to the framework of the analyser.

The connecting tube is held in place by the shape of the cups and the loose ground-glass joint (GGJ) which fits into the cup of the burette inlet tube, and by a rubber band passing from the edge of the framework of the analyser. It is steadied also by a split rubber collar (RC) which fits loosely around the neck of the collecting cup inside the outer cup.

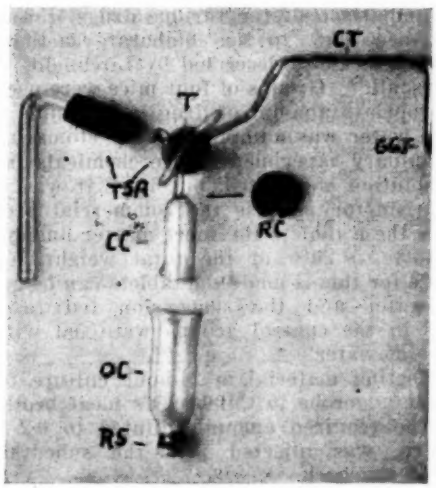

FIGURE VI.
Introduction apparatus for small volumes of gas, showing the various parts disconnected. Description of the apparatus is given in the text.

Both cups are filled with mercury. After an analysis, excess nitrogen is discharged from the analysing burette through the outlet and connecting tube into the collecting cup, followed by mercury until both burette and connecting tube are free of gas. While mercury is still running through, the tap on the connecting tube is locked. Suction is then applied to the side arm of the three-way tap and this tap is then turned to connect the side arm with the collecting cup. In this way the nitrogen is sucked from the collecting cup, this and the bore of the tap being filled with mercury. The tap is then locked.

Introduction of the sample from the tonometer without loss of volume is then carried out. A separate mercury reservoir with rubber tubing is attached to the side arm of the tonometer inlet, and the mercury in the inlet tube is adjusted to fill the bore completely. A hypodermic needle filled with glycerol is fitted to the inlet tube and the glycerol is forced out by mercury, the needle and inlet tube being left free from air. The needle is now driven from below through the rubber stopper in the bottom of the outer cup of the introduction apparatus, passing up within the collecting cup (Figure III). The upper tonometer tap is now opened to connect the tonometer with the needle. The sample is then forced out through the needle and rises into the collecting cup by allowing mercury to enter the tonometer from below. The needle is now withdrawn. The sample is readily drawn from the collecting cup into the analysing burette. Care must be taken to prevent any serum or blood from following the gas sample into the collecting cup, or if this should take place it must be prevented from being withdrawn into the connecting tube and burette, which may be accomplished by use of the tap above the collecting cup.

Another form of combined tonometer and introducer was tried, in which one inlet tube of the tonometer was lengthened and bent in two right angles ending in a ground joint to fit directly into the cup of the inlet tube of the analyser. The tonometer, however, was more cumbersome to handle and necessitated disconnexion from its reservoir during sampling.

An estimation of the accuracy of the whole technique of sampling, introduction and analysis was made in a manner similar to that described for the animal investigations, except that the volumes of the samples withdrawn from the reservoirs were kept within the range from 0·2 cubic centimetre to 2·0 cubic centimetres. Twenty-two consecutive samples were taken, the volumes being approximately as shown in Table I. The variations from control Haldane analyses are given as percentages and in millimetres of mercury partial pressure from averages of errors at each volume.

TABLE I.

Volume. (cubic centimetres.)	Percentage Variations from Control Analyses (Haldane).		Variations in Partial Pressure from Haldane Control Analyses. (Millimetres of mercury.)	
	CO_2.	O_2.	CO_2.	O_2.
2·0 1·5 1·0	−0·09	±0·04	−1	±1
0·6 0·45	−0·23	±0·11	−2	±1
0·3	−0·37	+0·16	−3	+2
0·2	−0·52	+0·26	−4	+2

The errors are caused by contamination by minute amounts of air in sampling and transference and by small pockets of nitrogen remaining in the tap of the analysing burette. Both these factors lower the carbon dioxide percentage as analysed, but tend to counteract each other as regards oxygen percentage. Gas tensions in depots in human subjects are given to the nearest millimetre of mercury. Unless otherwise indicated, all gas tensions given are calculated from samples of 0·5 cubic centimetre in volume or greater, so that variations of more than two millimetres of mercury are of significance.

It must be noted that the accuracy of the analysing apparatus used cannot be taken as the accuracy of the whole technique in any method of sampling. The errors from sampling are likely to be far greater than those of analysis.

Calculation of Results.

All gas compositions are given in terms of millimetres of mercury partial pressure. The total dry

gas pressure of the depot is taken as the corrected barometric pressure *minus* 47 millimetres of mercury (water vapour pressure at body temperature), and the partial pressures of carbon dioxide and oxygen are calculated from this in the usual way, the percentage figures given by analysis being used.

Summary.

A technique is described for the estimation of gas tensions in the tissues by the gas depot method, applicable to human and animal investigations. A modified sampling tonometer is used in which dead space is eliminated, and a method of introduction of gas samples into a gas microanalyser is given. The technique makes possible the collection of gas from the tissues and the analysis of large or small samples of gas without loss of volume.

Acknowledgements.

I am indebted to the Australian National Health and Medical Research Council for its generous grant, which has assisted the research in gas tensions in the tissues.

My personal thanks are due to Professor Lambie for his supervision and assistance in this work, and to Mr. M. Morrissey, of the Department of Medicine, for the photographs of apparatus published.

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A NOTE ON THE TREATMENT OF GAS GANGRENE WITH SULPHANILAMIDE AND RELATED COMPOUNDS.

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WITH the advent of another war, which will in all probability involve many of the medical problems of the Great War, it is not surprising that the question of sulphanilamide therapy for gas gangrene has been raised. Already Long and Bliss[1] have reported good results in *Clostridium welchii* infections both in man and in laboratory animals, whilst Domagk[2] has found that *Clostridium welchii* and *Clostridium septicum* infections in mice respond particularly well to sulphanilamide compounds with a substituted sulphamino group. As far as I am aware, the only unfavourable report is from Kendrick,[3] who, working with guinea-pigs infected with *Clostridium welchii*, failed to detect any therapeutic effects with "Neoprontosil", sulphanilamide or sulphapyridine.

Most of the investigations appear to have been carried out on *Clostridium welchii* infections, and in this preliminary report a short account is given of the effect of various sulphanilamide drugs on infections with *Clostridium welchii*, *Clostridium septicum* and *Clostridium œdematiens*. Other experiments with *Clostridium histolyticum* and the use of combined serum therapy and chemotherapy will be reported at a later date.

Methods.

For comparison of the various drugs it was not thought necessary to use elaborate methods of assay, such as those described by Litchfield, White and Marshall.[4] Groups of four mice were used and the appropriate amount of drug in 0·5 cubic centimetre of water was administered by stomach tube. In preliminary experiments pure chemicals in gum acacia solution were used, but later it was found quite satisfactory to use the commercial preparation. In these tablets the inert matter amounts to about 15% to 20% of the total weight, and if allowance for this is made the tablets can be ground up in water and the suspension fed to mice. Animals in the control groups were fed with 0·5 millilitre of water.

As infecting material, a 24-hour culture of the particular anaerobe in Colebrook's meat broth was used. The required amount, diluted to 0·2 cubic centimetre, was injected into the subcutaneous tissues of the back.

Preliminary tests showed that the best results are obtained if administration of the drug is commenced within two hours after infection and successive doses are given at frequent intervals.

In Table I the degrees of toxicity of the various sulphanilamide compounds are shown. The figures refer to the initial dose, which was repeated three times over twenty-four hours. The lowest amount of the drug, in milligrammes, that kills all four mice of the group is chosen as a lethal dose, and the toxic dose is defined as that which visibly affects all the mice. It is found that infected mice are slightly more susceptible to the toxic effects of sulphanilamide drugs.

TABLE I.

Lethal and Toxic Doses in Milligrammes for a Mouse Weighing 20 Grammes.

Dose.	Sulphanilamide.	"Prontosil rubrum."	"Uleron."	"M & B 693."	"Soluseptasine."
Lethal	45	50	—	—	—
Toxic	40	40	200	200	—
Tolerated ..	30	30	100	100	50

Infection with Clostridium Welchii.

Table II shows the therapeutic value of various sulphanilamide drugs in mice infected with one minimum lethal dose (0·015 cubic centimetre) of *Clostridium welchii*. Treatment consisted of four equal doses of the drug administered by stomach tube two, four, seven and ten hours after infection. Four groups of four mice were used for each test.

These experiments indicate that all the above drugs afford a moderately good protection against infection with one minimum lethal dose of *Clostridium welchii*. As is the case with streptococcal and pneumococcal infections, the best protection is given by drugs such as "M & B 693" or "Uleron", in which the sulphonamide group is substituted. Drugs in which substitution has been made in the amino group have the same or sometimes even a weaker action than sulphanilamide. If one expresses strength of action as chemotherapeutic indices, that is, the ratio between toxic and curative doses, these would be: for "Soluseptasine", 1:3; for sulphanilamide and "Prontosil rubrum", 1:6; and for "Uleron" and "M & B 693", 1:13.

Infection with Multiples of One Minimum Lethal Dose of Clostridium Welchii.

The therapeutic values of sulphanilamide and "M & B 693" were also tested in mice infected with multiples of one minimum lethal dose of *Clostridium welchii*. The results are shown in Table III.

As soon as multiples of one minimum lethal dose are used for infection, accurate interpretation of results becomes impossible. In every batch of mice some survive, but the majority cannot be cured. In these latter tests, apart from the anaerobic organisms, a greater quantity of toxin has been injected and therefore combined treatment with sulphanilamide and antitoxic serum would probably be much more efficient than treatment with sulphanilamide alone.

Infection with Clostridium Septicum.

Therapeutic tests carried out after the infection of mice with one minimum lethal dose (0·12 cubic centimetre) of *Clostridium septicum* gave the results shown in Table IV.

In infections with one minimum lethal dose of *Clostridium septicum*, the small doses of sulphanilamide drugs, which had been efficient with *Clostridium welchii*, proved valueless. Some effects comparable with those obtained with multiples of one minimum lethal dose of *Clostridium welchii* could be obtained with very high doses, which approached the limit of tolerance of infected mice. In this instance my experience differs from that of Domagk, who obtained good results in *Clostridium septicum* infections. It may be that different strains are sensitive to a different degree to the action of the drugs employed.

Infection with Clostridium Œdematiens.

The results of therapeutic tests carried out on mice infected with slightly less than one minimum

TABLE II.

Drug.	Group.	Dose. (Milligrammes.)	Fate of Mice.	Controls.
Paraaminosulphanilamide.	1	20·0	All 4 survived.	3 died (1 day). 1 died (2 days).
	2	10·0	All 4 survived.	
	3	5·0	All 4 survived.	
	4	2·5	3 died (1 day). 1 died (3 days).	
"Prontosil rubrum."	1	20·0	All 4 survived.	1 died (1 day). 2 died (2 days). 1 died (5 days).
	2	10·0	3 survived. 1 died from extraneous causes.	
	3	5·0	All 4 survived.	
	4	2·5	1 survived. 2 died (1 day). 1 died from extraneous causes.	
"Soluseptasine." (Administered by intramuscular injection.)	1	30·0	All 4 survived.	
	2	15·0	All 4 survived.	
	3	7·5	3 survived. 1 died (2 days).	
	4	3·5	1 survived. 2 died (1 day). 1 died (5 days).	
"Uleron."	1	20·0	All 4 survived.	3 died (1 day). 1 died (3 days).
	2	10·0	All 4 survived.	
	3	5·0	3 survived. 1 died (4 days).	
	4	2·5	1 survived. 2 died (1 day). 1 died (3 days).	
Sulphapyridine ("M & B 693")	1	20·0	3 survived. 1 died from extraneous causes.	
	2	10·0	All 4 survived.	
	3	5·0	All 4 survived.	
	4	2·5	1 survived. 1 died (3 days). 2 died (5 days).	

TABLE III.

Minimum Lethal Doses of *Clostridium welchii* in cubic centimetres.	Drug.	Group.	Dose. (Milligrammes.)	Fate of Mice.	Controls.
2 (0·03)	Sulphanilamide.	1	30·0	1 survived. 2 died (1 day). 1 died (2 days).	3 died (1 day). 1 died (2 days).
		2	20·0	2 survived. 2 died (1 day).	
		3	10·0	3 survived. 1 died (1 day).	
2 (0·03)	"M & B 693."	4	30·0	1 survived. 3 died (1 day).	
		5	20·0	3 survived. 1 died (3 days).	
		6	10·0	2 died (1 day). 1 died (2 days). 1 died (3 days).	
5 (0·07)	Sulphanilamide.	7	30·0	1 survived. 3 died (1 day).	4 died (1 day).
		8	20·0	4 died (1 day).	
5 (0·07)	"M & B 693."	9	30·0	1 survived. 3 died (1 day).	
		10	20·0	1 survived. 2 died (1 day). 1 died (3 days).	

TABLE IV.

Drug.	Group.	Dose. (Milligrammes.)	Fate of Mice.	Controls.
"M & B 693"	1	50·0	3 survived. 1 died (2 days).	1 died (1 day). 2 died (2 days). 1 died (3 days).
	2	50·0	3 survived. 1 died (2 days).	
"Uleron"	3	50·0	1 survived. 1 died (1 day). 2 died (2 days).	
Sulphanilamide	4	30·0	1 survived. 2 died (2 days). 1 died (5 days).	
"Prontosil"	5	30·0	1 survived. 2 died (1 day). 1 died from extraneous causes.	

lethal dose (0·0008 cubic centimetre) of *Clostridium œdematiens* are shown in Table V.

In this infection, attempted cure with sulphanilamide drugs met with complete failure. Not only was cure or prolongation of life impossible, but in some experiments, in which the lethal dose was not reached, the treated mice died sooner than the controls, and in some instances more of the treated than of the control mice died. It therefore appears that treatment of *Clostridium œdematiens* infection

TABLE V.

Drug.	Group.	Dose. (Milligrammes.)	Fate of Mice.	Controls.
"M & B 693"	1	40·0	4 died (1 day).	1 survived. 1 died (1 day). 2 died (2 days).
Sulphanilamide	2	20	1 survived. 2 died (1 day). 1 died (2 days).	
"Prontosil"	3	20·0	2 died (1 day). 2 died (2 days).	
"Uleron"	4	40·0	2 survived. 1 died (2 days). 1 died (3 days).	2 survived. 2 died (3 days).

TABLE VI.

Drug	Group.	Dose.	Fate of Mice.	*Post Mortem* Findings.	Controls.	*Post Mortem* Findings.
"M & B 693"	1	50 milligrammes.	1 survived.			
			1 died (1 day).	*Clostridium œdematiens*, coliform bacilli.		
			1 died (1 day).	*Clostridium tetani*, *Clostridium septique*.		
			1 died (3 days).	No organisms.		
	2	20 milligrammes.	1 died (3 days).	*Clostridium œdematiens*.		
			1 died (3 days).	*Clostridium septique*.	1 died (3 days).	*Clostridium œdematiens*.
			1 died (3 days).	*Clostridium septique*.		
			1 died (4 days).	*Clostridium welchii*, streptococci and *Clostridium septique*.	1 died (3 days).	*Clostridium œdematiens*.
	3	7·5 milligrammes.	1 died (3 days).	*Clostridium œdematiens* and coliform bacilli.	1 died (4 days).	*Clostridium welchii* and *Clostridium œdematiens*.
			1 died (4 days).	No organisms.	1 died (4 days).	*Clostridium œdematiens* and coliform bacilli.
			1 died (4 days).	*Clostridium septique*.		
			1 died (4 days).	*Clostridium œdematiens*.		
"Soluseptasine"	4	0·3 cubic centimetre 5% solution (intramuscular injection).	1 died (2 days)	*Clostridium œdematiens*, *Clostridium septique*.		
			1 died (2 days).	No organisms.		
			1 died (4 days).	*Clostridium welchii*.		
			1 died (4 days).	*Clostridium œdematiens*.		
Sulphanilamide	5	30 milligrammes.	1 survived.			
			1 died (1 day).	No organisms.	1 died (2 days).	*Clostridium septique*.
			1 died (3 days).	*Clostridium œdematiens*.		
			1 died (3 days).	*Clostridium septique*.	1 died (3 days).	*Clostridium septique*.
	6	20 milligrammes.	2 survived.			
			1 died (3 days).	No organisms.	1 died (3 days).	*Clostridium welchii* and *Clostridium septique*.
			1 died (3 days).	*Clostridium septique* and *Bacillus proteus*.		
"Uleron" ..	7	50 milligrammes.	1 died (2 days).	*Clostridium œdematiens*.	1 died from extraneous causes.	
			1 died (3 days).	*Clostridium œdematiens* and *tridium septique*.		
			1 died (3 days).	*Clostridium tetani*, *Clostridium œdematiens* and *Clostridium septique*.		
			1 died (4 days).	*Clostridium septique*.		

with sulphanilamides should not be attempted. Perhaps mixed infection with anaerobes not susceptible to the action of sulphanilamides is the reason for the failure sometimes encountered in the treatment of gas-gangrene infections in humans.

Infection with Garden Soil.

In some further experiments the various drugs were tested on infections produced with manured garden soil. The latter was sieved, dried in the incubator, and finally ground up in a glass mortar; 0·035 gramme of garden soil (one minimum lethal dose) was taken up in 0·2 cubic centimetre of normal saline solution and injected into mice in the subcutaneous tissues of the back. The results of these tests are shown in Table VI.

In view of the experiences with pure cultures of anaerobes it is not surprising that unfavourable results were obtained with sulphanilamide drugs on infections with soil containing all types of anaerobes.

Summary and Conclusions.

In experiments with pure cultures of *Clostridium welchii*, *Clostridium septicum* and *Clostridium œdematiens* and in infections with garden soil, the comparative values of sulphanilamide and two drugs with a substituted amino group, "Prontosil rubrum" and "Soluseptasine", and two with a substituted sulphamino group, "Uleron" and "M & B 693", were tested. As in other infections, the drugs with the substituted sulphamino group proved more efficacious in infections with pure cultures of *Clostridium welchii*. No definite protection could be obtained in infections with *Clostridium septicum*, and complete failure was encountered in infections with *Clostridium œdematiens*. Even at the best the action of sulphanilamide drugs in infections with pure cultures of *Clostridium welchii* must be designated as weak in comparison to the powerful action of the same drugs on streptococcal and other infections.

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Addendum.

Since this work was submitted for publication a paper has appeared by Stephenson and Ross in *The British Medical Journal*, March 23, 1940, page 471. Working on somewhat similar lines, they obtained results in agreement with those described in the present article.

Reviews.

ESSAYS IN PATHOLOGY.

MEDICAL men throughout Australia have learnt to welcome the appearance of articles from the pen of Dr. Reginald Webster. Readers of THE MEDICAL JOURNAL OF AUSTRALIA have been fortunate; for he has been a frequent contributor to its pages. His "Pathological Reports from

the Children's Hospital, Melbourne", which appeared serially in this journal during 1938 and 1939, have been collected into one small volume.[1] The result is worthy of Dr. Webster and the hospital. In the preface to the book Dr. Webster writes: "I have been actuated by a desire to focus attention on a number of interesting specimens which in the seclusion of a pathological museum would in the ordinary course of events come to the notice of comparatively few." These specimens are rare; some of them are almost freakish. Simple descriptions of them from a fluent pen would be of sufficient interest to make the book worth reading. But there is much more in this book than simple descriptions of unusual specimens. Every report is an essay with the smooth flow that we expect in Dr. Webster's writings. In every one there are philosophical considerations that are worthy of attention for themselves alone. And in every one there is something to make the thoughtful person think. Dr. Webster's questions are as interesting as his conclusions.

The preface concludes with these words: "The work involved for me will be amply rewarded should the book be conceded sufficient merit to bring a measure of credit to the Children's Hospital, Melbourne, an institution which induces a deep attachment in all connected with it." Dr. Webster can be assured of receiving much more than this modest reward.

The book is attractively produced. The printing is good. Art paper has been used throughout. The illustrations are excellent.

PROGRESS IN PHYSIOLOGY.

THE sixth edition of "Evans' Recent Advances in Physiology" has been revised by W. H. Newton.[2] The range of subjects reviewed is a wide one: the physiology of bone; the Carrel-Lindbergh perfusion apparatus; the sex hormones; the oxygen supply of the fœtus, rapid reactions in the transport of carbon dioxide; the metabolism of cardiac muscle; carbohydrate metabolism; secretion of urine; nervous control of micturition; chemical transmission at nerve endings; spinal reflexes; and the cortical control of muscular movements.

The sections on bone, the Carrel-Lindbergh apparatus, carbohydrate metabolism and cortical control of muscular movements are new, the other sections have been revised.

The reviews are all of a very high standard if somewhat specialized, but they require very close attention in reading. In a short review it is impossible to discuss the treatment of the varied subjects. The section on carbohydrate metabolism is particularly stimulating and thought-provoking and could, with advantage, be studied by all who have to do with derangements of carbohydrate metabolism.

The section on the Carrel-Lindbergh perfusion apparatus gives a short but clear account of this extraordinary apparatus and its use in keeping organs alive for days or even weeks. The section on the secretion of urine is an excellent summary of experimental evidence on the functions of the different parts of the kidneys with a discussion on the substances used for plasma clearance tests. The other sections are more purely physiological.

When we consider the many thousands of papers on physiology and related subjects which appear every year, this book must be regarded as a very successful attempt to present the current ideas on a large section of physiology.

THE DIAGNOSIS OF SWELLINGS.

A BOOK of 300 pages by C. E. Corrigan, Lecturer in Surgery, University of Manitoba, entitled "The Clinical Diagnosis of Swellings",[1] is divided into two sections. The former deals with the differential diagnosis of swellings by their characters and apart from their regional status; the latter is devoted to the problems of diagnosis of swellings in well-known regions. The reader may be surprised to find included a chapter on "Ulcers", but the reason for this inclusion is given by the author and appears to be justified.

The section devoted to hydatid cysts is brief and the conclusion given that "as a rule, diagnosis by clinical means alone is seldom conclusive" will hardly be appreciated by Australian readers.

The statement that in inguinal hernia the "commonest content is ileum, the bladder occasionally is present, the Fallopian tube more rarely so" takes no cognizance of the frequency with which colon and omentum are found in the sac.

The author does not claim to cover the whole field of surgery, but does give in a condensed and concrete form much valuable information and useful hints compiled from various sources. At times reference is made to conditions which one hardly expects to find in modern surgery and which are to be anticipated only in neglected cases. The text is illustrated by line-drawings or diagrams, and these will be found to be very practical in elucidation of the subject matter in the different sections.

The book is one which sums up much that the student has learned during his medical curriculum; it would be useful particularly for reference by resident medical officers in large institutions, or by practitioners when confronted by problems of diagnosis. The text is clear, the subject matter is concise and a useful index is appended.

A BOOK ON DIET.

"DIET IN HEALTH AND DISEASE", a hand-book published on behalf of *The Practitioner* and edited by Sir Humphry Rolleston and Dr. Alan Moncrieff, is a good book.[2] It merits the praise given by Professor Cathcart in the introduction. In 331 pages thirty authors contribute twenty-eight chapters in a comprehensive survey of the subject of diet.

Food is treated from the viewpoint of the scientific laboratory, the kitchen, the sick patient and the convalescent. Throughout there is a leavening of common sense and of the practical. The articles are interestingly written and make enjoyable reading for medical men and (in the case of the less technical chapters) for laymen.

The scope of the book may be judged by a random selection of the chapter captions. "Practical Dietetics", by Professor V. H. Mottram, comprises nineteen pages of interesting data concerning "not only what foods are, but what they do!"—the vitamins, the daily rations and the list of "protective foods". It is noted that the available iron value of spinach and meat is dethroned and the cheapness and availability of iron in black treacle (9·17 milligrammes per 100 grammes) is stressed. Chapter II. "Sick Room Menus and Recipes", is by R. H. Wansbrough; Chapter IV, "Dispensing Special Diets", is by M. Abrahams. Other chapters deal with diet in gastric diseases, diet in *diabetes mellitus*, diet in disorders of the cardio-vascular system, diet in allergic diseases, diet in rheumatism and gout, diet in old age, general principles of diet in childhood, artificial feeding of infants, special diets in childhood and diet in convalescence. A chapter on the effect of cooking on food, by A. B. Callow and E. M. Widdowson, is very interesting and special attention is given to the effects of cooking on vitamins.

The hand-book may be recommended; it is well printed on good paper, well indexed and a credit to the publishers.

[1] "Pathological Reports from the Children's Hospital, Melbourne", by R. Webster, M.D., D.Sc., F.R.A.C.P.; 1939. Sydney: Australasian Medical Publishing Company, Limited. Medium 8vo, pp. 165, with 38 illustrations. Price: 8s. 6d. net.

[2] "Evans' Recent Advances in Physiology"; Sixth Edition, revised by W. H. Newton, M.D., M.Sc.; 1939. London: J. and A. Churchill Limited. Large crown 8vo, pp. 501, with 109 illustrations. Price: 15s. net.

[1] "The Clinical Diagnosis of Swellings", by C. E. Corrigan, B.A., M.D., F.R.C.S.; 1939. London: Baillière, Tindall and Cox. Medium 8vo, pp. 321, with illustrations. Price: 18s. net.

[2] "The Practitioner Handbooks. Diet in Health and Disease", edited by H. Rolleston, Bt., G.C.V.O., K.C.B., M.D., F.R.C.P., and A. A. Moncrieff, M.D., F.R.C.P.; 1939. London: Eyre and Spottiswoode Limited (for The Practitioner). Demy 8vo, pp. 382. Price: 14s. net.

The Medical Journal of Australia

SATURDAY, JUNE 8, 1940.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE PREVENTION OF BLINDNESS.

BLINDNESS and the blind may be considered from several points of view. The attitude of most, and possibly of all, people is humanitarian—sympathy and pity are aroused at the misfortune, the deprivation and the sorrow of a brother man, and not seldom a wondering admiration is compelled by the bravery and cheerfulness with which he overcomes his disabilities. But this attitude does not exclude, since it often gives rise to, other standpoints. The social economist sees in blindness an economic loss to the individual and to the State, and is concerned with measures, such as education and vocational training, that will enable the blind to earn a living and take their share in the life of the community. Those whose interest lies in preventive medicine study the causes of blindness that they may prevent its occurrence. Though it is imperative that everything possible should be done to lessen the hardships of the blind and to enable them to become self-respecting and self-supporting members of society, the preventive point of view is beyond question the most important of those that have been mentioned; and it is unfortunately true that sufficient attention is not being paid to it in Australia at the present time.

Any community which sets out to prevent blindness among its members must have experts who are prepared to discover and to study the causes of blindness. It must then determine whether any of these causes can be removed or prevented from exercising their influence. When this has been done, and only then, is it possible to institute preventive measures. In countries such as England and America organizations dealing with blindness in all its aspects have been established; but workers in these countries have recognized that in addition to the essentials already named it is necessary to discover the extent and the incidence of blindness in the community and to know what ocular lesions are responsible for it. No one will deny that it is at least sensible to know the magnitude of a task before it is undertaken. In an important work, "Blindness and the Blind in the United States", Harry Best reproduces many tables setting out the percentage distribution. He has taken his figures from reports of the United States Census Bureau, and estimates that about 72% of the blindness in the United States is preventible. The percentage for a country like Australia cannot be very different. Unfortunately we have no exact data on which to base such an estimation, apart from a survey that comes from Tasmania. A report of this work, carried out by Dr. J. Bruce Hamilton and Dr. W. D. Counsell, was published by the National Health and Medical Research Council with the report of its meeting held at Hobart in February, 1937. A supplementary report was published in this journal in March, 1939. The Tasmanian investigators concluded that at least 40% of the blindness in Tasmania was preventible; and they stated as the three main causes, heredity, injury and venereal disease.

To consider the causes of blindness as they affect man in his several stages from birth to old age is not sufficient; the antenatal period and hereditary influences have to be taken into account. The most important hereditary diseases of the eye are

retinitis pigmentosa and hereditary optic atrophy. If it is known that persons intending to marry have a bad family history, the attending ophthalmologist will naturally give the appropriate advice. Unfortunately family histories are not always known, and when information of this kind is sought, the greatest difficulty is sometimes experienced. Much more research on this subject is needed, apart from the plotting of genealogical trees in affected families. We may state in passing that some important work in the last-named subject is at present being carried out at Hobart. In regard to the antenatal period, there is no need to lay stress on the necessity for efficient treatment of the mother if she should be affected by either syphilis or gonorrhœa. *Ophthalmia neonatorum* was the disease that received most attention when the Society for the Prevention of Blindness was established in Great Britain as far back as 1880. Karl Credé, a German gynæcologist, introduced the prophylactic instillation of silver nitrate drops into the conjunctival sac of new-born infants in 1881, and this one procedure has been more effective than any other in reducing the incidence of blindness. E. K. Macdonald has recently stated that whereas in 1907 40% of blindness was due to *ophthalmia neonatorum,* the percentage in 1939 was less than one. Hamilton and Counsell found that seven of 92 cases of blindness in Tasmania were due to *ophthalmia neonatorum.* In the pre-school period blindness or serious loss of sight may result from untreated strabismus, progressive and untreated myopia and certain sequelæ of infectious diseases. At the school age myopia presents the greatest problem. It should perhaps be noted at this stage that we have not given a definition of blindness—a matter of some difficulty, for it does not always indicate total loss of vision. Sir James Barrett has discussed the subject on more than one occasion, and his views have been published in this journal; in the space available the question cannot be further pursued at present. Neither need extensive reference be made to special measures for sight saving at schools, since this was done in the issue of February 24, 1940, in reference to the sight-saving school recently started at Hobart by the Tasmanian Department of Education. Attention may, however, be drawn to a prophecy made by E. K. Macdonald, medical officer of health of Leicester, in addressing a conference on "blind welfare" at Birmingham, England, on June 29, 1939. Macdonald said that it was not unreasonable to prophesy that at a not too distant time it would be the custom for every child to have a full refractive examination of his eyes when he entered school, with a full examination of his fundi and media by a competent ophthalmologist. This is an objective to be borne in mind by all ophthalmologists who are interested in the prevention of ocular disability. In adult life accident is possibly the most prolific cause of loss of sight. Modern factory regulations are framed to give protection to workers; and, apart from carelessness, there is little risk of injury in the ordinary course of events. Still, accidents do occur and eyes are injured; and in this regard reference has to be made to efficient lighting. Bad lighting is not only conducive to accident, but has an effect that is far from good on eyesight and on the general comfort of the worker. This applies to schools and offices as well as to factories. Of the cataract of old age little need be written except to emphasize the desirability of operation when perception of light and projection are good and when there is no disease in the other eye. Hamilton and Counsell made a point in their report that will surprise many people. They hold that when a patient has a cataract that is suitable for removal, he should not be granted a pension if he has refused to submit to operation. They pointed out that the blind person who is helpless and dependent is very much under the domination of his relatives, to whom, by being blind and in receipt of a pension, he is an asset; being restored to sight, he may only be a liability. "Whereas the patient may be only too glad to have his vision restored by operation, his decision is formed by others." That such conditions may exist is deplorable; and the views expressed by Hamilton and Counsell should at least receive consideration by those controlling the pensions regulations.

No mention has been made of war blindness; this is in itself a large subject and must be left

for another occasion. Apart from this, it must be clear from what has been written that much can be done to prevent loss of sight and that much remains to be done in Australia. Research has to be carried out; surveys have to be made; the activities of existing agencies have to be extended and coordinated; and knowledge at present available, but not used, must be applied. In all this work trained ophthalmic surgeons should take a leading part, for they alone are qualified to express an opinion on this important subject. The problem is Australian-wide and should absorb the attention of the Ophthalmological Society of Australia (British Medical Association). Sir James Barrett, in his president's address to the first annual meeting of the society, suggested that the prevention of blindness should be one of its main activities. The society has drawn up a scheme showing what research is necessary and has submitted it to the National Health and Medical Research Council. If the council is not prepared to authorize research in this subject, it might, on the public health side of its activities, try through the State departments to give effect to the recommendations made to it in 1937 by Hamilton and Counsell.

Current Comment.

LOW SOIL PHOSPHATE AND AUSTRALIAN MILK.

THAT Australian soils have a low concentration of phosphates has been known for some considerable time. Victorian clay soils have approximately one-third of the phosphorus percentage found in similar soils in the United States of America, and indeed the concentration is below what American agriculturists regard as necessary for successful production. In the coastal regions of New South Wales the amount is so small that one wonders how plant life is as healthy as it is. Hilda Kincaid[1] in 1911 showed that indigenous Australian vegetation has attuned itself to phosphate dearth, whereas imported grasses and cereals have a struggle to obtain their customary quantity and generally fall a little below success, as is very demonstrable in Australian wheat. . Before superphosphate was employed as a top-dressing, the depletion of phosphates from the grass lands of Australia continued at an alarming rate and threatened eventual sterility of the soil. The export of carcasses of sheep, oxen and rabbits, of milk products and bone meant a steady drainage of the weakest constituent of the land, whilst added to actual export was the loss through rabbits eating grass on the uplands but dying in the gullies, where the calcium phosphate of their skeletons could not enrich the soil. Despite the great importation of mineral fertilizers, the productive land in Australia has still a concentration of available phosphate below what is found in Europe and America.

The very important question now arises whether this phosphate deficiency affects Australian nutrition. A distinguished English biochemist suggested that the high incidence of dental caries in Australia could be referred to this soil characterestic, but this hypothesis can be countered by the argument that if the phosphate deficiency is severe enough to affect the teeth, it would also affect the bones; but the stature and general physique of Australian youth negative this assumption.

A laborious analysis of Australian milk by R. C. Hutchinson[1] gives results which are most interesting. Many expected to find the phosphorus percentage of Australian milk well below normal; but this is not the case. To give actual figures, the average phosphorus percentage of milk in Europe and America is 0·091, whereas that of Australian milk is 0·082; in other words, the average Australian milk contains 90% of the amount of phosphorus found in the average milk of Europe and America and is well within normal limits. The one disability discovered by Hutchinson is that a correlation exists between the vitamin *A* content of butter fat and the phosphate concentration of the fodder, and as this latter is below European and American averages there is a small reduction in the vitamin *A* content. The figures cited by Hutchinson give the average phosphorus percentage of mixed pasture in Britain as 0·3, whereas the results of his analyses of Australian pastures varied from 0·149 to 0·178. That the lower percentage of vitamin *A* could be responsible for any malnutrition is most unlikely. Between the other vitamins and the mineral ingredients of milk no correlation was found, other factors being dominant. The general outcome of this extended research is that Australian milk ranks high in Calorie yielding ingredients, and, as we know, in vitamin *D*. On the other hand, as stated, the phosphate and vitamin *A* content are a little lower than British and American averages, but are within normal limits and can be regarded as quite satisfactory; indeed it may be claimed that few cities and towns in other parts of the world are supplied with milk of the Australian standard.

It will be remembered that Hutchinson published in this journal last September an article embodying the results of a research on the nutritive value of powdered milk of Australian manufacture.[2] Chemi-

[1] *Proceedings of the Royal Society of Victoria*, Volume XXIII (N.S.), 1911.

[1] *Proceedings of the Royal Society of Victoria*, Volume LII, 1940, page 113.
[2] THE MEDICAL JOURNAL OF AUSTRALIA, Volume II, 1939, page 392.

cally and biologically tested, the powdered milk came out of the comparison rather well, being actually richer in utilizable iron and copper than ordinary milk. Cats given no other food lived a little longer on the fresh milk than on the reconstituted milk made from the powder; the fat content of the latter was slightly lower; but with these exceptions there was no detectable difference between the two. Judged by bacterial purity, the powdered was easily ahead of the fresh milk. Hutchinson's admirably restrained summing up states that: "Although the reconstituted product did not replace fresh cow's milk it was an excellent substitute and in some of its properties was even superior to it." This is welcome news to those who live in the tropics and to others who may through various causes be temporarily deprived of fresh milk. The results of Hutchinson's further nutritional researches will be awaited with interest.

THE MENACE OF MARIHUANA.

AMERICA has a new drug problem. Hashish, the drug of dreams that added spice to the hedonism of other ages, has come back under the Mexican name of marihuana to add new notes to the neurotic's repertoire and fresh sensations to the pleasure-seeker's palate. Throughout the vast continent of America marihuana smoking has, according to recent authorities, assumed almost epidemic proportions. It is smoked indiscriminately by the old and the young. It is peddled publicly and has found its way far beyond the vice dens of large cities into schools and orphanages. It has a vernacular of its own; and the public conscience has recently awakened to the fact that many school children smoke "mootas" and "reefers" and indulge in "laughing jags".

Marihuana is a product of hemp (*Cannabis sativa*), a plant which is cultivated in parts of the United States for its fibre, and which grows wild throughout large tracts of the country. The dried flowering tops of this plant are rolled into cigarettes and sold in drug and grocery stores, night clubs and other pleasure resorts. Legend obscures the origin of the hashish vice; but English and French literature contain vivid descriptions of its seductiveness. Narcotics have always fascinated individuals who desire to escape from reality through some temporarily pleasurable alteration of the personality.

Marihuana smoking produces a variety of sensuous experiences. A pleasant, though peculiar, sense of unreality develops soon after the smoke is inhaled. Later, a state of "double consciousness" may be felt, in which the subject fancies he sees himself in the delirium but is unable or unwilling to leave it. Time and space are singularly distorted, and complicated hallucinations of sight and hearing may become manifest. Vivid and highly coloured dreams float through the mind; and states of rapturous ecstasy may alternate with periods of terrifying apprehension. Laughter may be uncontrollable. The smoker generally feels stimulated and may dance and sing, with a feeling of pleasant euphoria. Obstacles seem to be surmounted with ease. Nothing is difficult; and the smoker feels inspired with confidence and possessed with the attributes of genius.

The psychiatric effects of marihuana have been studied by Bromberg,[1] who bases his views on a study of 42 patients suffering from mental disorder and admitted to hospital following the use of marihuana. Bromberg believes that marihuana smoking may induce an acute intoxication or a toxic psychosis. The symptoms of intoxication last from a few hours to several days and clear up without any sequelæ. The toxic psychoses may persist for many weeks or even months; and the toxic picture is frequently superimposed upon an underlying psychotic disturbance, such as schizophrenia. Many persons who use marihuana exhibit no mental symptoms. But over-indulgence is easy, and the symptoms of an acute delirium may necessitate the smoker's removal to a mental hospital. Unlike morphine, marihuana apparently produces no withdrawal symptoms. The symptoms of delirium include increased psychomotor activity, rapid and incoherent speech, disorientation, excitement, which is often sexual, and the presence of hallucinations. The subjective symptoms include feelings of intellectual brilliance, the sensation of walking on air, and various somatic feelings, including lengthening of the limbs. Acts of violence may be committed and impulsive suicidal attempts have been recorded. The psychiatric reactions to marihuana are dependent on the personality make-up of the patient. Very often the addiction commences in the incipient stages of a functional psychosis, before the final breakdown of the adaptive mechanism. As addiction proceeds, toxic symptoms develop: disorientation, agitation, hallucinations and clouding of consciousness appear in the foreground of the clinical setting and may persist for some weeks. Then, as the toxic symptoms recede, a typical schizophrenic or manic-depressive picture is revealed.

Bromberg doubts whether marihuana is habit-forming; but other observers disagree with him on this point and believe that many users of marihuana require to take larger doses. In a number of cases it would seem that habituation to marihuana has led to heroin or morphine addiction; for in a survey of 58 heroin addicts it was discovered that 44% began by smoking marihuana. Drug pedlars have been known to adulterate marihuana cigarettes with heroin in order to augment their nefarious trade. In this lies the chief menace of marihuana. The hemp narcotics do not provide that sustained retreat from reality which is the Nirvana of certain psychopathic personalities. They provide only temporary shelter for those who are overburdened with inferiorities or fretted with minor discontents. To many who crave a new sensation, marihuana offers

[1] *The Journal of the American Medical Association*, July 1, 1939.

perilous though transient delights. To those whose conflicts are more deep-seated, marihuana is merely a stepping stone to morphine and all the misery that its addiction entails.

VITAMIN K.

OF the newer vitamins that are now being used therapeutically, vitamin *K* is of quite recent application and promises to be one that will be especially valuable. Vitamin *K* is a fat-soluble vitamin, present in large amounts in certain green vegetables, notably lucerne and spinach. Its existence was first suspected by Dam in Copenhagen, when, in some studies in lipoid metabolism, newly hatched chicks placed on a fat-free diet developed a tendency to hæmorrhage. Further studies showed that a new fat-soluble vitamin is essential for the prevention of this bleeding tendency. The substance was designated vitamin *K*, the letter standing for the Dutch word *Koagulation*. Subsequent work by Dam and his colleagues[1] has shown that a deficiency in vitamin *K* results in a lowered blood prothrombin level, which, when reduced to levels below one-third of normal, gives rise to spontaneous hæmorrhage. After injury severe hæmorrhage may occur owing to interference with the clotting time of the blood, and sufferers behave in many respects like hæmophiliacs. The vitamin can be produced by bacterial action and is therefore present in the lower portion of the intestine, even when the animal is maintained on a diet free of the vitamin. Absorption from the lower part of the intestine is minimal in the chick, but does occur in mammals. Hence mammals, including man, rarely show vitamin *K* deficiency except in cases of faulty absorption. But as the absorption of fats from the bowel is largely dependent on the action of bile, it follows that a deficiency of bile such as occurs in obstructive jaundice, biliary fistula and degeneration of the liver, will hinder or prevent the absorption of fat-soluble vitamins, including vitamin *K*. After a few weeks or months the plasma prothrombin level falls and a tendency to bleeding develops. Treatment of the hæmorrhagic tendency consists in feeding bile or bile salts along with large amounts of vitamin *K* to supplement the natural supply.

H. P. Smith and his co-workers, S. E. Ziffren, C. A. Owen and G. R. Hoffman, have stated[2] that vitamin *K* deficiency should be suspected in all cases of biliary fistula or obstructive jaundice. It is well known that in operations on the gall-bladder and bile ducts severe hæmorrhage is a frequent complication. Consequently, if a deficiency does exist, it is important that the patient receive vitamin *K* therapy for several days before any operative procedures are taken. On the other hand, this routine treatment in all suspected cases will result in needless delay when vitamin *K* deficiency has not yet developed. Smith has therefore described a new test for bedside detection of deficiency.

Deficiency of vitamin *K* also provides an explanation of hæmorrhagic disease in the newly born. A full-term infant usually has only one-sixth to one-third of the adult proportion of prothrombin, and premature infants have much less. In the most deficient babies the condition becomes manifest as a hæmorrhagic state. The low prothrombin levels in infants can be raised by feeding vitamin *K* to the mother before delivery or to the infant directly after birth. In infants with *icterus gravis* or *anæmia neonatorum*, the tendency to hæmorrhage (but not the anæmia or jaundice) may be relieved by vitamin *K*.

Clinically active vitamin *K* has been obtained principally from putrefied fish meal and alfalfa meal. The preparation and purification of crystalline products have been announced almost simultaneously in many different laboratories. It is now generally agreed that the vitamin contains a quinone structure, and it seems that vitamin *K* activity may be a property of several quinone derivatives. Thus S. Ansbacher and E. Fernholz have found[1] that 2-methyl-1 : 4-naphthoquinone is practically as active as vitamin *K*; while Fieser and his colleagues, from experiments on day-old chicks, suggest[2] that some of the 2-hydroxy-3-alkyl-1,4-naphthoquinones possess positive vitamin *K* activity. J. Al. Macfie and his co-workers[3] have described the clinical use of 2-methyl-1 : 4-naphthoquinone as a synthetic substitute for natural vitamin *K*. The results reported showed the synthetic substance to be superior to natural vitamin *K* concentrates in raising the prothrombin index.

Various phases of the chemical, physiological and biological aspects and the clinical usefulness of vitamin *K* are developing so rapidly that a number of current views may require modification in a very short time. However, the importance of anticipating the danger of prothrombin deficiency and detecting it by means of laboratory studies will remain a paramount consideration in the successful treatment of hæmorrhagic tendency.

[1] *Nature*, April 27, 1935, page 653; *The Biochemical Journal*, June, 1936, page 1075; *ibidem*, March, 1938, page 485.
[2] *The Journal of the American Medical Association*, July 29, 1939, page 380.

WIRELESS BROADCAST OF AN OPERATION.

IN *Time*, of March 25, 1940, appears the following extraordinary story:

> Under a hot lamp in the green-tiled operating room, the patient lay masked and draped in white. Only a six-inch patch of iodine-stained abdomen was exposed. Although numbed below the waist by a spinal anæsthetic, he could hear everything that was going on. Only unusual fixture in the room was a microphone . . . linked to another, held behind the glass observation window by Announcer Bill Sandiford of Mutual's Station KOL. Said he: "Ladies and gentlemen, you are about to hear the first broadcast of an actual operation right now at King's County Hospital in Seattle . . . [under the sponsorship] of the Washington State Medical Association."

[1] *Journal of the American Chemical Society*, July, 1939, page 1924.
[2] *Ibidem*, page 1929.
[3] *The British Medical Journal*, December 23, 1939, page 1220.

Abstracts from Current Medical Literature.

RADIOLOGY.

Barium Rubber Drains.

GEORGE M. WYATT (*Radiology*, November, 1939) suggests that no rubber articles should be introduced into natural or artificial body cavities unless they can be demonstrated by X rays if the need arises. Barium sulphate was selected for the opaque ingredient because it was found to give the greatest density with the least alteration of the physical properties of rubber; at the same time it is chemically inert. Thin rubber drains containing 25% barium sulphate by weight were readily demonstrable through the abdomen of an obese patient. To avoid confusion of these drains with others in the operating room a brilliant scarlet pigment (chlornitraniline red) of the same type as that used in the manufacture of toy balloons was used. With advances in processes of manufacture, particularly in the field of synthetic textiles, all other materials used in surgical procedures may eventually be made radio-opaque.

Intraspinal Lesions Associated with Low Back Pain and Sciatic Pain.

JOHN D. CAMP AND E. A. ADDINGTON (*Radiology*, December, 1939) admit that lipiodol is an irritant to the spinal meninges, and emphasize that its use is contraindicated in frank inflammatory lesions. Nevertheless, they state, the consensus of experienced neurologists and neurosurgeons is that, when it is used judiciously and in selected cases, the advantages of its use far outweigh any disadvantages. Experiences with its use in the recognition of space-occupying lesions affecting the spinal cord and *cauda equina* indicate that this procedure has attained a degree of accuracy that is equalled by few other diagnostic radiological methods. Within the last two years there has been a tendency to revive the use of air as a contrast agent for the localization of lesions within the spinal canal, and the statement has been made that the use of lipiodol in obstructing lesions and in lesions affecting the *cauda equina* is unjustifiable. There is no doubt that air injections will reveal certain of these lesions, and undoubtedly they should be used more frequently before resort is had to lipiodol. The authors' experience to date, however, with both methods in regard to lesions involving the lumbar portion of the spinal canal, indicates that lipiodol is more accurate, that it will reveal certain structures not seen with air, and that it has localized lesions that air has failed to disclose. Because of the obvious difficulty in controlling the position of air in the cervical and thoracic regions, there is no comparison in the efficiency of the two methods when these regions are involved, except perhaps in the case of a completely obstructing lesion.

Intestinal Obstruction.

N. S. ZEITLIN (*Radiology*, November, 1939) stresses the importance of technically good films with elimination of all movement in cases of intestinal obstruction. Whenever possible the patient should be moved to the X-ray table and high milliamperage should be used to cut down the time of exposure to a fraction of a second. When this is impossible, the Lysholm grid should be used. Multiple views in prone and supine positions are frequently necessary. A negative diagnosis based on a single film, in a case of suspected obstruction, may lead to error. Gas in the small bowel is not sufficient to warrant a diagnosis of obstruction; distension is essential. The relative size of the colon and the small bowel must always be remembered. Frequently the obstruction will manifest itself by a visualized distension of only one isolated loop of small bowel. The passage of small amounts of stool material and gas does not rule out the diagnosis of intestinal obstruction.

The Conservative Treatment of Intussusception in Children.

JENS NORDENTOFT (*Acta Radiologica*, April, 1939) advocates the reduction of large intestinal intussusceptions—colic and ileo-colic forms—by barium enema when they come under treatment in the first twenty-four hours. In nearly all cases reduction can be accomplished. It is most important to obtain sufficient pressure with the enema, and the column of barium should be at least forty inches high, unless, of course, the reduction has been effected before this pressure has been reached. In early cases even higher pressure may be used without any risk of rupture of the intestine. Reduction is indicated by disappearance of the swelling and copious discharge of fæces with the enema, or by spontaneous backflow into the small intestine. If the reduction is not certain, a very light anæsthetic should be given while the child still retains the enema. In a large majority of cases this will result in outflow into the small intestine. Although a good outflow into the small intestine from a well-filled caecum gives considerable certainty of reduction, it is possible that a fresh invagination may occur from a slight remaining infolding in the caecum.

X-Ray Diagnosis of Hæmangioma of Long Bones.

M. J. NEMENOV (*Radiology*, October, 1939) states that when a hæmangioma involves the long bones, the bones are thickened and the structure of both the sponge-like substance and the compact layer is greatly changed. The bone appears to have a nuclear structure, the latter sometimes spreading to the very periosteum. However, the periosteum is hardly ever impaired and the tumour does not penetrate into the adjoining tissues. Nor does the tumour grow into the epiphyseal cartilage or penetrate into the joints. In flat bones as well as in other bones having a large diameter (as the head of the femur) it is possible to see that the osseous substance forms a centre from which the osseous plates spread radially, resembling a colony of the ray fungus. The nuclei formed by the osseous beams are usually small in size; but sometimes they may be larger and resemble the picture usually presented by a giant-cell tumour or echinococcus infestation of the bone. However, in the case of the hæmangioma the larger nuclei are in their turn penetrated with a fine loop network of osseous substance. Besides, in the case of a giant-cell tumour, the bone appears to be inflated and always has a cortical layer, however fine. In hæmangioma, as previously mentioned, the nuclei reach as far as the periosteum.

The "Cut-off of the Diaphragm Line" in Radiographs of the Chest.

FRANCIS POLGAR (*Acta Radiologica*, June, 1939) describes a new sign which helps to determine the cause of small areas of opacity occurring in the right costo-phrenic angle. These opacities may be due to consolidation in the lower lobe of the right lung or to a small pleural effusion or pleural thickening. The contour of the right side of the diaphragm, as seen in the X-ray film, may be divided into two parts. The medial two-thirds are in contact with the middle lobe and the outer third is covered by the antero-lateral portion of the lower lobe. If a shadow formed in the right costo-phrenic angle is caused by a marginally demarcated area of increased density of the lower lobe, the diaphragm contour is abruptly cut off at a basal anterior point where middle and lower lobes meet. The author calls this sign "the cut-off of the diaphragm line". If the right costo-phrenic angle is filled with pleural fluid, the medial edge of the shadow will present quite a different picture at the place where it meets the diaphragm line. The medial inferior end point of the shadow of an effusion can lie medial or lateral to the interlobar fissure, but, unlike a consolidation, it seldom coincides exactly with the lobar margin. Furthermore, when the shadow is due to an effusion, a blurred and hardly definable medial border is found, owing to the fluid extending along between the diaphragm and the base of the lung. If the shadow is caused by consolidation of the lower lobe, the end point of the free diaphragmatic contour will lie anteriorly on the anterior edge of the fissure. The cut-off is therefore located closely

behind the frontal thoracic wall at a point which can be demonstrated by rotating the patient. Small effusions will, as a rule, lie more posteriorly.

PHYSICAL THERAPY.

X-Ray Treatment of Hyperthyreoidism.

G. E. PFAHLER (*Radiology*, January, 1940) has had thirty-five years' experience of the irradiation treatment of hyperthyreoidism. He believes that irradiation is indicated in all cases of hyperthyreoidism in which the patient is not in crisis or is not suffering from definite pressure symptoms. He states that the advantages of irradiation are as follows: (i) The fear of operation is eliminated and therefore the patient is more likely to come under treatment early and before cardiac damage has taken place. (ii) If the patients are treated reasonably early, they are not interrupted in their occupation. (iii) There is no pain or shock and no great inconvenience results if the condition is treated reasonably early. (iv) Patients with advanced disease or serious heart complications may be treated without shock. (v) There is no risk of death from the treatment. (vi) There is an absence of scar or keloid formation. (vii) The end results are approximately equal to those obtained by surgery. The various objections usually offered to X-ray treatment are discussed in detail. The danger of burns does not arise in modern radiotherapy of this disease. Exacerbation of symptoms after irradiation has occurred rarely in the author's cases, and in his experience is insignificant and no more than may occur from time to time independently of treatment. Myxœdema has not occurred, and hypothyreoidism in a mild form only very rarely. Clinical proof of the absence of danger of damaging the thyreoid cells or parathyreoids is shown by the fact that with the enormous amount of irradiation given in the treatment of cancer of the larynx no case of hypothyreoidism or of tetany is recorded in the literature. Difficulty in subsequent surgical treatment has been adduced as an objection to irradiation. It is pointed out first that in only a small percentage of cases submitted to irradiation is subsequent operation necessary. Further, it is now considered that adhesions found at operation cannot be attributed to irradiation when it has been given along modern lines. Slowness of response to irradiation has been objected to; but this cannot be said to be a serious objection if the patient is treated reasonably early. The question of permanent cardiac impairment developing during prolonged irradiation treatment must be considered; but it must be remembered that the fear of operation leads to delay, and if irradiation is considered the patient will be treated earlier, before serious heart damage has taken place. Details of technique are given, and reference is made to the treatment of hyperthyreoidism by irradiation of the pituitary gland as practised by Borak and others. The author is convinced that it is inadvisable to use iodine in conjunction with X-ray therapy. Of the patients treated from 1933 to 1937 he claims that 62% have been cured, 33% considerably improved, and only 5% not improved. It is stated that irradiation is probably of value in the prevention of the development of carcinoma in toxic adenomata.

Radiological Treatment of Carcinoma of the Œsophagus.

R. B. ENGELSTADT (*Acta Radiologica*, September, 1939) states that the results obtained in the treatment of carcinoma of the œsophagus have so far been very poor, as a permanent cure has only rarely been observed. The histological structure of these tumours is, on the other hand, of such a kind that *a priori* we may assume that they are radiosensitive. During the years 1932-1938, 119 patients suffering from œsophageal carcinoma have been seen at the Norwegian Radium Hospital. Of these, 111 have been treated by irradiation; but the treatment has in 33 cases been quite incomplete. Biopsy was done in 83 cases. It is difficult in certain cases to obtain a satisfactory biopsy, and this happened in this series 15 times, when the diagnosis was clinically and radiologically that of carcinoma. The primary results are that four patients are symptom-free, the condition of 21 is highly improved, that of 32 is not improved, and in 16 instances the condition was progressing during treatment. Patients have been put down as symptom-free only when X-ray examination and œsophagoscopy revealed a complete disappearance of the tumour for a shorter or longer period of time. Post-mortem examination was performed in eight cases, but in two of these no treatment had been carried out or treatment was incomplete. Of the other six cases no trace of the carcinoma was found in one, great effect of the irradiation in two, and a moderate effect in three cases. If possible, combined radium and deep X-ray treatment is given. The radium is placed in a sound in the œsophagus and a total of 30 Dominici units (corresponding to about 2,500 *r*) is given. Four fields are used in the deep X-ray therapy, with daily doses of 150 to 300 *r*, and a total of 12,000 *r* or more. With combined treatment the maximum depth dose obtained on the inside of the œsophagus may be 5,500 *r*. To complete the treatment eight to ten weeks are required. The combined radium and deep X-ray treatment with heavy doses seems to give better results than deep X-ray treatment alone. Heavy doses are necessary. The local and general reactions from the irradiation must be carefully treated. Nourishment must be as abundant and adequate as possible. The author is under the impression that the value of gastrostomy is very limited.

Small Dose Irradiation in Functional Gynæcological Conditions.

I. I. KAPLAN (*American Journal of Roentgenology*, November, 1939) states that since the physiological response of body functions to irradiation has been better understood, Röntgen and radium therapy are employed to control various endocrine dyscrasias and dysfunction of vital organs. In many women who are desirous of bearing children, when all other efforts have failed to bring about this result, Röntgen therapy in many instances has proved effective. Amenorrhœa, when due to functional disturbances, is also amenable to Röntgen therapy. The author reports the results in the treatment of 194 married women who suffered from persistent amenorrhœa and sterility. All the patients had previously been examined for gynæcological abnormalities. In all instances some form of endocrine therapy had been used and found unavailing. In many histories previous tubal insufflation was recorded, and in a large number of instances the husband had been examined for potency. In a number of cases a previous surgical corrective operation or curettage was carried out without relief of symptoms. In all cases irradiation with high voltage X-ray therapy was administered in small doses to the ovaries. In many of the cases additional X-ray therapy was given to the pituitary and in a few instances to the thyreoid gland. Of 194 patients treated, 159 were followed up, and among these menstruation was reestablished in 103. Of the 103 patients, 51 became pregnant. Reports from the parents have in no instance disclosed any abnormalities or physical deformities in the children. The oldest child was twelve years of age when the article was written. There still exists a prejudice in the profession against irradiation for therapeutic purposes because of alleged uncontrollable harmful effects upon both mother and offspring. This is due to a misconception, for while X rays may be harmful to the embryo *in utero*, this action is entirely different from that upon unimpregnated ova. Irradiation properly given in small doses, as in the series reported, is in no way harmful to either mother or offspring. Detailed reports of many of the cases are given. The exact explanation of the action of X rays in this type of case is not definitely known, although several theories have been formed. Whether the effect is on the ovary, uterus, the pituitary or some indefinite endocrine factor is debatable.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at the Women's Hospital, Carlton, Melbourne, on April 17, 1940. The meeting took the form of a series of clinical demonstrations by members of the honorary medical staff of the hospital. Part of this report appeared in the issue of June 1, 1940.

Jaundice.

DR. ARTHUR HILL reported a series of twenty cases of jaundice among patients who had been in hospital during the previous fifteen months. In four cases the jaundice was associated with pregnancy at or near term. These four cases were of the following types: one was a case of catarrhal jaundice; one was a case of severe anæmia of pregnancy simulating the Addisonian type, and responding to blood transfusions and liver therapy; one was a case of acute yellow atrophy of the liver following vomiting in the later months of pregnancy, and proving fatal some hours after expulsion of a macerated fœtus; and one was a case of acute infective endocarditis due to infection with *Streptococcus hæmolyticus*, group A. The last case, which closely resembled an infection due to *Bacillus welchii*, had recently been reported in THE MEDICAL JOURNAL OF AUSTRALIA. There were sixteen cases of jaundice following abortion, the majority the result of criminal interference. Of these, twelve were due to infection with *Bacillus welchii*, three to lead poisoning and one to a leptospiral infection.

The twelve cases due to infection with *Bacillus welchii* were all of a grave type; ten of the patients died, one within fifteen minutes of her admission to hospital. All showed the characteristic muddy or orange jaundice, with rapid deepening and underlying cyanosis, attaining in some a bronze hue. The urine, when obtainable, was scanty and strongly acid; in eight cases it was of a burgundy or port-wine colour, or black, owing to the products of hæmolysis it contained; in one case it contained urobilin, but no blood pigments; and in three cases it was either unobtainable or its qualities were not recorded. In those cases in which the blood serum was examined, this also was of a burgundy colour, on account of hæmolysis. In addition to the routine intensive administration of fluids, antitoxin and alkalis, with blood transfusions when indicated, seven of these patients had an immediate hysterectomy performed; two survived. Of those who died after operation, one succumbed to a reactionary hæmorrhage, one to pulmonary embolism on the sixth post-operative day, and one who died on the eighth post-operative day was also extensively infected with *Streptococcus viridans*. In all twelve cases *Bacillus welchii* was recovered from the patient during life, and the organisms were cultured from the vagina, uterus, peritoneal cavity, urine and blood of one survivor.

The three patients with lead poisoning, which had appeared to result from swallowing diachylon plaster, were not seriously ill. All complained of abdominal colic, and showed a typical lemon-yellow jaundice, with pallor, a blue line on the gums, and basophilic stippling or polychromasia in the blood film. Constipation was not invariable.

The one patient with a leptospiral infection, whose case had been more fully reported by Dr. J. W. Johnstone, died on the seventh day after her admission to hospital. For over twenty-four hours the clinical course closely resembled that of infection with *Bacillus welchii*. In addition to muddy jaundice and a prostrating toxæmia, the patient showed petechial hæmorrhages on the tip of the nose, a feature confined in Dr. Hill's experience to severe infection with *Bacillus welchii*, and invariably accompanied by gross evidences of hæmolysis in the urine. In this case, however, the urine was free of blood pigments, and the later clinical course was not that of severe infection due to *Bacillus welchii*. Bacteriology played the important role in this case at the outset in excluding *Bacillus welchii* as a cause, and later in establishing the true nature of the infection.

Normocytic Anæmia of the Puerperium.

DR. J. D. HICKS showed a *primipara*, aged thirty-three years, suffering from severe normocytic anæmia of the puerperium. As her temperature was over 100° F. on four successive days her puerperium was classed as morbid. No severe blood loss was observed during or after labour. No blood examinations were performed until the patient was noticed to be pale during the second week of the puerperium. At that stage her hæmoglobin value was 34%, the erythrocytes numbered 1,360,000 per cubic millimetre, the colour index was 1·25, and the leucocytes numbered 3,450 per cubic millimetre. Examination of the film revealed a fair degree of anisocytosis with many microcytes and some cells which appeared larger than normal. Three erythroblasts were seen when 100 leucocytes were counted. A week later the hæmoglobin value had fallen to 24% and the erythrocytes had correspondingly fallen in number to 1,150,000 per cubic millimetre, the colour index to 1·04 and the leucocytes to 2,600 per cubic millimetre. There was no response to an intramuscular injection of "Campolon". The highest reticulocyte count was 1·6% four days after the injection. The patient was given several transfusions of blood, with corresponding improvement, but she was still very ill, as she had some degree of septicæmia. The appearance of the early films suggested a macrocytic anæmia of the liver-deficiency type; but as the patient showed no response to liver therapy and subsequent blood examinations revealed a colour index only slightly above 1·0, it was believed that she was suffering from normocytic anæmia.

Hæmoglobin Estimations in Pregnancy.

Dr. Hicks also showed the results of 400 hæmoglobin estimations of patients at various stages of the pregnancy. The hæmoglobin percentage of the average patient attending antenatal clinics in the early months of pregnancy was 10% to 20% below the normal average for the healthy non-pregnant female, as computed from groups of female students and nurses. During the later months of pregnancy the hæmoglobin percentage gradually dropped 10% to 15% below that found in the first three months of pregnancy.

Panhysterectomy for Bacillus Welchii Infection.

DR. JOHN GREEN showed a patient who had had panhysterectomy performed for *Bacillus welchii* infection. The infection had developed in connexion with therapeutic abortion, induced by the tube method, in the fifth month of pregnancy. The indication for termination was grave toxæmia in previous pregnancies—albuminuria with blindness in the seventh pregnancy and eclampsia followed by still-birth in the eighth pregnancy. Induction by the tube method had been performed on a Friday; by Sunday the patient was having rigors and on Monday afternoon she was seen by Dr. Green. She was very ill and drowsy, jaundiced and vomiting greyish-green fluid. There was a dark offensive discharge from the vagina, but the uterus had not emptied itself. The urine was dark reddish-brown in colour. Her temperature was 99·4° F. and her pulse rate 104 per minute.

As the patient still had some "circulation" left (the extremities were warm and the pulse, though thin, was not unduly rapid) and the uterus had not emptied itself, immediate operation was arranged. At operation the uterus and appendages were removed by panhysterectomy, the anæsthetic being ether and oxygen. After the operation the patient was given gas gangrene antiserum, and in the first five days 450,000 units were either given intramuscularly or added to the glucose and saline solution that was given intravenously. Except for a severe rigor the morning after operation the patient made an uninterrupted recovery. Cultures of material from the

vagina, uterus and urine yielded a growth of *Bacillus welchii*, *Bacillus coli communis* and a non-hæmolytic streptococcus; no organisms were grown on attempted culture from the blood. The bacteriological investigation was carried out subsequent to the operation.

Dr. Green said that the following points called for comment: (i) the risk of infection with the tubal method of induction; (ii) the difficulty of emptying the uterus halfway through the pregnancy (abdominal hysterotomy and sterilization would probably have been a more satisfactory procedure); (iii) the fact that the decision to operate was made on the clinical picture. Septicæmia following tubal induction, with jaundice and dark urine, suggested *Bacillus welchii* infection. The circulatory condition of the patient had not degenerated so much as to make operation a grave risk and recovery improbable.

Hirsutism.

Dr. Green presented the results of investigation of a case of pronounced hirsutism. The patient, who could not attend the meeting, was aged twenty-eight years and unmarried. For two years, and particularly in the past twelve months, she had grown an obvious dark beard and moustache; the pubic hair was abundant and tended to a male distribution. At the same time the interval between the menstrual periods had lengthened to two months, although the loss was normal. She was a fresh-complexioned, heavy-looking woman, slow of speech and action. There was no history of abnormal sexual activity and the external genitals were not abnormally developed. The breasts were fairly well developed. Vaginal examination under anæsthetic disclosed that the ovaries were enlarged to the size of walnuts; the uterus was normal in size, with a cavity of eleven centimetres in diameter. The urine gave a slight reaction with Fehling's solution and a glucose tolerance test suggested renal glycosuria. The basal metabolic rate was – 10%. An X-ray examination of the pituitary fossa disclosed no abnormality. An investigation of the androgens in the urine was carried out by Dr. Vera Krieger, and the results indicated that a large amount of androgens was present. An X-ray examination after perirenal insufflation suggested that the upper pole of the left kidney was a little bulbous compared with the right side, but the appearance was not characteristic of a tumour. The patient appeared to be suffering from hirsutism, with an excess of androgens in the urine, but no gross virilism. The ovaries were enlarged and there was an equivocal appearance in the X-ray picture of the left kidney. In the circumstances the patient was given the option of an exploratory operation. At operation cystic areas were resected from each ovary and the knife was then handed over to the urologist, Dr. Harold Moore. Through a left-sided Kocher type of incision the kidney was exposed, but the suprarenal gland appeared normal. The right suprarenal area was palpated, but not seen. In view of the present incomplete knowledge it was not considered desirable to resect any of the suprarenal gland. The ovarian tissue contained numerous follicular cysts, but no sign of any masculinizing tumour. There had been no obvious change in the patient's condition, although the androgen content of the urine was now normal.

(To be continued.)

Medical Societies.

MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING of the Medical Sciences Club of South Australia was held at the University of Adelaide on December 1, 1939.

Demyelination of the Monkey Brain.

PROFESSOR E. WESTON HURST spoke on "Experimental Demyelination of the Monkey Brain". The characteristics of the demyelinating diseases of man and animals were outlined briefly. Professor Hurst said that a condition resembling in some respects Schilder's encephalitis in man had been produced in monkeys by repeated doses of potassium cyanide. He described and demonstrated the clinical and histological features of the experimental condition.

MR. I. W. MCDONALD discussed the general features of enzootic ataxia, a disease of lambs, which occurs commonly in South Australia. The major feature of the pathology was demyelination of certain tracts in the white matter of the spinal cord and of the white matter in the brain, and it had been shown that a dietary deficiency of copper was the cause of the disease.

In discussing Professor Hurst's paper, MR. R. MARSTON outlined briefly our present knowledge of copper metabolism. He said that the so-called polyphenol oxidases were known to be copper compounds and that the enzyme responsible for the reoxidation of the cytochrome-c in the normal cycle of oxygen utilization by living tissues was either a copper complex or a complex dependent on copper for its formation. The concentration of cytochrome-c and that of cytochrome-c oxidase was decreased in the tissues of animals that had been rendered copper-deficient by depletion of this element in their diet.

Apart from the general impairment of iron metabolism, which resulted in hyperchromic anæmia and hæmosiderosis of the liver and other organs, the most striking effect of copper deficiency in the sheep was the incidence among them of spastic paralysis, particularly of the hind limbs, which was the result of lesions of diffuse symmetrical demyelination of the white matter of the cord and cerebrum. The incidence was highest in lambs during the first few weeks after birth.

The lesions arising from chronic copper deficiency, with the resulting impairment of the cytochrome-c oxidase system, resembled those arising from chronic hydrogen cyanide poisoning sufficiently to suggest a common origin. Cyanide was well known to poison irreversibly the cytochrome-cytochrome oxidase system. Copper deficiency was known to impair the production of cytochrome and its associated oxidase. The observed demyelination of cerebral white matter then might possibly be due in either case to the particular sensitivity of the myelin to oxygen want; if so, the cytochrome-cytochrome oxidase system was not unlikely to be the site of impairment. Mr. Marston stated that such speculations should be capable of experimental verification.

Public Health.

POLIOMYELITIS IN MELBOURNE.

A REPORT on the poliomyelitis epidemic of 1937 and 1938 as it affected the city of Melbourne has been prepared by Dr. Hilda Bull, medical officer of the Department of Health of the Melbourne City Council, and is published under the authority of the Health Committee of the Council.[1] It is stated in the foreword that the survey and the clinical and epidemiological work on which the report is based were carried out under the direction of Dr. John Dale, Medical Officer of Health to the city of Melbourne.

The circumstances surrounding the outbreak of poliomyelitis in the winter of 1937 were unusual. The weather was characterized by "low temperatures, absence of rain and wind, and the most severe frosts and fogs ever recorded in Melbourne", and all the unusual features were accentuated at the time when the virus is believed to have been brought to Australia from New Zealand, in

[1] "Poliomyelitis in the City of Melbourne 1937-8", by Hilda W. Bull, B.Sc., M.B., B.S., D.P.H., Medical Officer, Department of Health, Melbourne City Council. Published under the authority of the Health Committee of the Melbourne City Council.

June. Moreover, there had been a much lower prevalence of other infectious diseases than usual; only mild rubella can be said to have had any prevalence at all. "It was not, therefore, into a community weakened by disease; but one that had been unusually free from the common ills, that the virus of poliomyelitis was introduced . . ."

The first cases of the epidemic occurred in the south-eastern suburbs in July. The outbreak was concentrated in a small district. It was realized at once that there were several "unusual and startling features". The disease was very severe, and it was obvious that a virus of much greater infectivity than that usually associated with the virus of poliomyelitis was at work. Moreover, owing to the work of the district health officer, Dr. C. R. Merrilees, in almost every case that occurred in the next district affected it was possible to establish a history of contact with one of the earlier cases.

> It became apparent that isolation of contacts, and segregation of susceptibles might be of paramount importance in limiting and slowing down the progress of the epidemic. Through the Press and the Radio the public was warned of the danger, and advised to adopt measures of isolation for all young children. A Consultative Council was appointed by the Government to advise the Chief Health Officer, Dr. H. N. Featonby, and to plan the campaign. One of their first actions was to recommend the "proclamation" of the earliest suburbs affected. In these areas schools were closed; mingling in crowds as at picture shows, and the movement of children to other areas were discouraged in every possible way. In spite of these precautions the epidemic "jumped" to the northern suburbs, and by the beginning of August, we in the City, who had been waiting anxiously to see what would happen, felt that a spread to our own district was inevitable. It was not expected that the industrial and poorer suburbs would suffer as severely; but we asked medical practitioners, schools and Health Centres in our own district to notify us immediately they heard of any cases of suspicious illness.
>
> As soon as a case was notified, the premises were visited, and the medical officer took a history of the symptoms and all concomitant events. All immediate contacts were medically examined and isolated. Among these a number of cases and abortive infections were discovered, and in many instances the probable infecting source was traced. Leaflets describing the symptoms and the measures of quarantine to be followed were distributed, and instructions given that all cases of ill-health in contacts were to be immediately reported to the Health Department. When these reports came in we visited the cases and kept them under observation. More remote contacts were visited by a staff of health inspectors, who reported any case of suspicious illness for immediate medical examination. For those illnesses considered to be due to invasions of the virus, strict quarantine was insisted on; but only those patients who showed some paresis or from the definiteness and severity of their symptoms seemed likely to be paralytic cases, were sent to hospital.

The first two cases in the city occurred on August 7. Both were at first "doubtful"; the first suggested rheumatism and the second meningitis. Within ten days "cases" and "abortives" had been reported from all over the districts in which these two patients lived. (For the purposes of the report, Dr. Bull defines the use of the words "case" and "abortive" as follows: ". . . the word 'case' will be taken to mean a paralytic case, and 'abortive', an infection which did not go on to paralysis. Only 'cases' were notified".) Parents became greatly alarmed, and an effort was made to prevent them from sending children to the country, lest the disease should be spread; but many did leave the city. An incidental result of the state of general apprehension was the financial loss suffered by picture shows and shops. "For a little while the streets where children usually played were deserted; but in our crowded areas they were soon back in the only playing space available to them." The "highest peak" of incidence in the city was reached in the fourth week of August, 1937, when there were 20 "cases" (6 fatal) and 27 "abortives". The total number of cases in the city for the whole epidemic was 174; 104 occurred in the first two months, and 9 of the 12 fatal cases also occurred in this period. There were apparently four "foci" in the city, chief of which was the out-patient department at the Children's Hospital. It was impossible to keep people away from the hospital, even by the offer of free medical attention. The other "foci" were "abortives" in districts far apart, and the whole of the city was involved within a few weeks. In spite of the prophylactic measures taken, the city epidemic did not end until March, 1938.

The baleful results of the epidemic were the worst on record. There were twelve deaths, and many other patients had to fight hard for life; the respirators and the special measures taken to treat glosso-pharyngeal paralysis must have prevented the mortality rate from being much higher. It was actually surprisingly low, being only 7%. In only six out of 174 "cases" did recovery without some paralysis occur. Preparalytic cases had been notified in previous epidemics; in this epidemic they were not notified, but were recorded as "abortives". This fact makes it difficult to draw any accurate comparison between this and other epidemics. If a distinction is made between paralytic and non-paralytic (notifiable) cases and "abortives", the total number of cases would be 168 paralytic, 6 non-paralytic and 199 "abortives". Dr. Bull states that if the same method of reporting cases were used as in other countries, at least half the "abortives" would possibly have been classed among the non-paralytic cases. This would add at least 100 to the total number of cases.

A total of 152 patients were left with some paralysis and required after-treatment. After they had been discharged from hospital (mostly wearing splints) they came under the supervision of the medical officers of the State After-Care Committee in charge of the "northern" district; Melbourne represented only a small part of this district. The first medical officer was Dame Jean Macnamara, and later Dr. Elizabeth McComas held the position. The most severely paralysed children were accommodated in large resident institutions; others were taken in ambulances each day from their homes to "all-day" clinics and returned later; others again, who could be treated at home, were visited by physiotherapists. The results of treatment are as follows: 36 of the 152 patients recovered after treatment lasting from three to twelve months; 101 were still being treated when the report went to press. (The report was written just over a year after the conclusion of the epidemic.) In 50 of these cases there is a good prospect of recovery; in 7 cases slight permanent disability seems likely, in 17 moderate disability will probably be left, and in 27 there will very likely be severe permanent disability. Of the severely paralysed patients, "some will be totally incapacitated, others will have to wear collars or callipers or have one or more limbs that are likely to recover very little function, and one is still in the respirator". Fifteen patients "discontinued treatment at their own risk"; they all had some paralysis before their defection from treatment, but on the whole it was not severe. The parents "became impatient at the immobilization"; and a few of the parents have returned "very much sadder and wiser, often with damage to the child which will take a long time to repair". The plight of one of these children is described:

> One of the children who was withdrawn from treatment, is practically paralysed from head to foot; but the parents took some comfort in the fact that he was "happier" out of his splints. However, they confess that deformities of the back and limbs are developing, and there seems no doubt that eventually he will require institutional care.

The general results of the epidemic are summed up in the closing paragraph of the report:

> The percentage of moderate or severe permanent damage is 28·5 per cent. When we consider also that 12 died, it is seen that the figures represent a considerable amount of suffering and deprivation, and are a tragic illustration of the ravages of this disease.

DR. G. M. OXER.

The severity of symptoms was not always a guide to the ultimate course of the disease. The abortive infection was of interest; it is believed that the early recognition of these infections may have helped to prevent the occurrence of paralysis in some cases. Many of the children who became paralysed had suffered from the biphasic or "dromedary" type of the disease and had been allowed to run about during the quiescent period. Therefore it was considered that complete rest "for some time" after the appearance of the first symptoms might help to prevent the development of paralysis. An interesting consideration is the possible effect of unusual activity on the occurrence of paralysis; this had been observed in previous epidemics, and it seems possible that fatigue may affect the outcome. This observation may also be of interest in connexion with the higher incidence of the disease in boys, who are naturally more active and enterprising than girls.

The cases were divided into two groups for purposes of classification. Group I consisted of notified cases of poliomyelitis in which paralysis or paresis was present; Group II consisted of unnotified cases of abortive poliomyelitis, subdivided again into (*a*) definite poliomyelitis and (*b*) probable infection. The cases in Group II presented difficulty in diagnosis; when paralysis occurred later the patients were transferred from the "abortive" to the "notified" list; but it was not until some months had elapsed that the distinction could be accurately made.

The epidemic was remarkable on account of four characteristics: (i) the large number of cases which were shown to be due to direct contact; (ii) the exceptionally virulent nature of the infection (the strain of virus involved appears to have been responsible for ten times the amount of "recognizable" illness that is usual); (iii) the large number of cases of upper cord and bulbopontine involvement; (iv) the occurrence of an epidemic of such "devastating proportions" in winter.

The first two notified cases occurred on August 7, 1937; the "peak" was reached by the end of August, and thereafter the epidemic gradually declined during September, October, November and December; from then until the end of March, 1938, only 18 cases occurred. Clinical observations confirmed the view of Dr. F. M. Burnet that the virus was entirely neurotropic and did not cause a systemic infection; it was therefore agreed that the use of convalescent serum was unjustified. This made the performance of lumbar puncture and examination of the cerebro-spinal fluid unnecessary, especially as it was required as an aid to diagnosis in only 30 cases in hospital. Lumbar puncture was not performed in the home.

> The character and course of the symptoms were, in the main, uniform and progressive, and the only difference was that in the abortive infections the disease was arrested before there was any considerable or permanent damage to the cells of the brain and spinal cord, particularly those of the anterior horns, whereas in paralysed and fatal cases such damage did occur.

Dr. Bull herself observed the symptoms in about 25% of cases and all "abortives"; in other cases they were elicited from the parents and medical attendant. This meant that some of the less conspicuous, but highly diagnostic, signs would be overlooked in some cases.

The most common signs and symptoms were the following: (i) elevation of temperature, not as a rule over 102° F.; (ii) headache; (iii) drowsiness; (iv) nausea and vomiting; (v) anorexia; (vi) pain in the back and neck; (vii) "flushing and paling"; (viii) sore throat; (ix) tremor; (x) head lag; (xi) a rapid pulse rate, from 120 to 140 per minute; (xii) constipation (not recorded as frequently as would be expected, because of the parents' recourse to castor oil); (xiii) mild and usually transitory strabismus and occasionally temporary blindness, photophobia and diplopia; (xiv) otorrhœa, in three cases, and "pounding" in the ears, in one case; (xv) sweating, in a few cases; (xvi) delirium; (xvii) epistaxis, in some cases and "abortives". The "dromedary" type of poliomyelitis was quite common; in this type mild symptoms first occurred, then there was a latent period of one to five days, after which the symptoms recurred in a much more severe form. Poliomyelitis was associated with other diseases in only a few instances.

There were 174 cases in the city of Melbourne; 82% of patients were under the age of ten years and 42% were under the age of five years; 56% were male and 44% were female; the 12 deaths were evenly divided between male and female patients. In the city population the incidence of poliomyelitis was 187 per 100,000; the incidence was lower in the adjacent and outer suburbs, the age incidence was slightly higher, and a rather greater proportion of males were affected. The incidence in the city of Melbourne was "the highest but one for the 'inner suburbs'". With regard to the municipalities, the highest incidence was in those where the population was dense and living conditions were rather poor. There were two exceptions; Essendon and Footscray, which are classed as "predominantly prosperous residential suburbs" had a high incidence, while Richmond, a "relatively crowded area", had a low incidence. It is suggested that this may be due to "a greater prevalence of the virus in this area some time during the recent past". Exposure to the risk of infection plays a large part, and isolation is difficult in poor and crowded areas. Immunization due to "silent" or "missed" infections in previous epidemics may determine the local incidence; there is no real proof either way, but support is lent to the idea by the greater incidence in the lower age groups in crowded communities.

There were 199 "abortives"; 87 (44%) of the patients were boys and 112 (56%) were girls. Often those who suffered from an abortive infection were much more ill than those who were paralysed; yet only five of those with abortive infections later had any paralysis, and it was very mild. There were a number of reports that paralysis, especially bulbar paralysis, followed tonsillectomy; the performance of tonsillectomy was postponed "by general consent" as soon as "the nature of the epidemic was recognized".

An interesting point raised in the report is the question whether "missed" infections are in any way responsible for some of the common orthopædic conditions, such as winged scapulæ, scoliosis and abnormalities of the feet and ankles. Several children who had had abortive infections were later found to be suffering from one or other of these conditions. It is considered that their occurrence may be purely coincidental.

A peculiar feature of "abortives" was that they gave rise to a "case" and an "abortive" in contacts; this feature was diagnostic of an abortive infection. The abortive infections were graded into three groups, A, B and C. The patients in group A were typically ill with poliomyelitis, and it seemed from the clinical appearances that paralysis might result. The patients in group B were not so ill, but it appeared that they had been infected with the virus of poliomyelitis. In group C the symptoms were milder still; but all the patients were contacts of "definite paralytic cases", and their symptoms appeared "within the limits of the incubation period, or simultaneously with the reported case". When the illnesses were simultaneous, in ten cases the source of infection was a previous "abortive".

> If, as there is good reason to think, abortive cases occurred among non-contacts in the same proportion as they did among contacts, we could postulate another 70 or 80 abortive cases among the general population of which we never heard. Even this is probably a conservative estimate.

The age incidence of "abortives" is a little later than that of "cases".

Observations made in this epidemic did not uphold the belief that the disease attacked chiefly the healthy, robust, well-cared-for children living in uncrowded suburbs. Actually, the overcrowded areas where undernourishment was prevalent suffered rather more than areas that enjoyed better conditions of living. The chief factors involved appear to be: (i) the impossibility of isolating children effectively; (ii) the fact that the families in these areas had an unsatisfactory standard of living (most were subsisting on sustenance or casual labour). Six cases occurred in institutions, and only 23 children (13%) were

living in fairly comfortable circumstances. The epidemic caused great hardship among the families involved, because a wage-earner from a home in which a case had occurred, although not officially quarantined, "often found his presence unwelcome at his place of employment, and was forced to stay away". Adolescent wage-earners had, of course, to be quarantined; and patients returned from hospital, often in splints, and required much attention, so that wage-earning mothers had to leave their employment to look after the child. This meant still less adequate food for the patient and for the rest of the family. Relief was arranged through the Ladies' Benevolent Society, and the work was approved and continued by the Health Committee.

The usual incubation period, judged as accurately as possible, appears to have been from seven to ten days; it was calculated as "the number of days elapsing between the first appearance of symptoms in the presumed infecting and secondary cases". In families the incubation period tended to be shorter.

> It is possible . . . that a case may be infectious before any signs appear, or the incubation period may be shortened where there is a greater opportunity for massive infection, as these children would mostly be living in crowded houses and often sleeping in the same bed.

Dr. Bull states that she is inclined to regard as due to simultaneous infection those cases in which symptoms appeared before the fifth day after they had appeared in the primary case; when more than twelve days elapsed after contact before the appearance of symptoms, she regards the infection as of the second generation, "probably through an intermediary with a mild or 'silent' infection".

With regard to the mode of spread of the disease, it is stated in the report that in most cases it was possible to trace some connexion with a previous "case" or "abortive". The question of "convalescent carriage" is considered; and though no absolute proof was obtained, it appears to have been responsible for several cases. There was no suggestion that the disease was spread by other than human contact. The question of closing schools had not been considered in earlier epidemics; but in the early part of this epidemic it was obvious that school contacts were responsible for a large number of infections. The closing of the schools to the younger children appears to have had considerable effect in reducing the spread of the epidemic, and it is believed that complete closure of the schools may have prevented the occurrence of a large number of cases and slowed the spread of the disease; but it is not suggested that this measure would have been "entirely effective".

The incidence of multiple infections in families was unusually high; 24% of families involved had more than one infection, either "cases" or "abortives".

> The occurrence of multiple cases in crowded homes, with a virus of high virulence, is, of course, to be expected. But it would be difficult to say whether it is only because, once the virus is introduced, the chances of its spreading are very great. The large number of simultaneous infections, i.e., occurring within 4 days of each other, however, suggests that there may have been in some instances a predisposition to infection by this particular virus in certain families.

There were no second attacks among the few children who had previously had poliomyelitis; but the brother and sister of a former patient had severe abortive infections. The general conclusion drawn from the Melbourne epidemic is that the position of the affected child in the family was of no significance.

Dr. Bull is to be congratulated on this comprehensive report; it should be studied by all who are engaged in the control of infectious diseases, and also by general practitioners, who are in this, as in so many other assaults on the public health, the first line of defence.

Post-Graduate Work.

COURSE IN ANATOMY AND PHYSIOLOGY.

AN eleven weeks' course suitable for candidates for Part I of the examination for the Degree of Master of Surgery has been arranged by the New South Wales Post-Graduate Committee in Medicine, and will be held at the University of Sydney, beginning on Tuesday, July 30, 1940. The course will end on Friday, November 1, 1940, but there will be a three weeks' vacation at one stage of the course, the dates of which will be announced later.

The lecturers will be as follows:

Neurology:
Professor A. N. Burkitt.
Dr. G. Phillips.

Anatomy:
Dr. F. A. Maguire (female pelvis).
Dr. A. A. Abbie (extremities).
Dr. I. D. Miller (head and neck).
Dr. F. W. Niesche (thorax and abdomen).
Dr. N. R. Wyndham (embryology).

Physiology:
Professor H. W. Davies (respiration and blood).
Dr. F. S. Cotton (cardio-vascular).
Dr. H. G. McQuiggin (endocrines).
Dr. N. R. Wyndham (biophysics and special senses).

Biochemistry:
Dr. H. S. H. Wardlaw.

The fee for attendance at the full course is twenty guineas, but parts only of the course may be taken, the fees for attendance at which are as under:

Physiology only (24 lectures)	£6	6	0
Biochemistry, all subjects (10 hours lectures and 20 hours practical)	10	10	0
Physiology and biochemistry (34 lectures)	12	12	0
Anatomy only (36 lectures)	8	8	0
Embryology only (8 lectures)	3	3	0
Neurology only (10 lectures)	4	4	0
Anatomy, all subjects (54 lectures)	12	12	0

Applications for registration, accompanied by a remittance for the amount of the fee, must be made to the Secretary, New South Wales Post-Graduate Committee in Medicine, the Prince Henry Hospital, Little Bay, from whom full copies of the programme may be obtained.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 92, of May 23, 1940.

AUSTRALIAN MILITARY FORCES.

NORTHERN COMMAND.

First Military District.

Australian Army Medical Corps.

Honorary Captain L. P. Sapsford is appointed from the Reserve of Officers (A.A.M.C.) and to be Captain (provisionally), 8th April, 1940.

Australian Army Medical Corps Reserve.

To be Honorary Captain—Abraham Fryberg, 6th April, 1940.

To be Honorary Captains—Kevin Walter Priddis, Roy Ira Mannion and Frederick Ernest Bright, 30th April, 1940. The resignation of Honorary Captain H. A. Sundstrup of his commission is accepted.

EASTERN COMMAND.

Second Military District.

Australian Army Medical Corps.

Major M. S. S. Earlam is seconded for Staff duties, 1st April, 1940.

Australian Army Medical Corps Reserve.

To be Honorary Captain—Charles William Sutherland Dun, 22nd April, 1940. Captain W. M. Amphlett is placed upon the Retired List with permission to retain his rank and wear the prescribed uniform. Honorary Captain A. M. Burge and Honorary Lieutenant S. H. Rogers are retired.

SOUTHERN COMMAND.

Fourth Military District.

Australian Army Medical Corps.

Honorary Captains K. T. Cooper and D. C. Dawkins are appointed from the Reserve of Officers (A.A.M.C.) and to be Captains (provisionally), 17th April, 1940.

Captain E. F. Gartrell is appointed from the Reserve of Officers (A.A.M.C.), and is borne supernumerary to establishment pending absorption, 3rd May, 1940.

WESTERN COMMAND.

Fifth Military District.

Australian Army Medical Corps.

Honorary Captain K. H. Tepper is appointed from the Reserve of Officers (A.A.M.C.) and to be Captain (provisionally), 4th April, 1940.

Australian Army Medical Corps Reserve.

To be Honorary Captain—Frank Edward Gallash, 17th April, 1940.—(Ex. Min. No. 98—Approved 22nd May, 1940.)

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force.

Medical Branch.

The following are granted commissions on probation with the rank of Flight Lieutenant, with effect from 26th April, 1940: Gordon Stirling Colvin, M.B., B.S., and Edward Wolf Field, M.B., B.S.

The notification in the *Commonwealth of Australia Gazette*, dated 24th April, 1940, concerning Patrick Francis Peter Ryan is cancelled.

Reserve.

Medical Branch.

John Herald Balfour Brown, M.C., O.B.E., is granted a commission on probation with the rank of Flight Lieutenant and the honorary rank of Squadron Leader, with effect from 13th May, 1940.

The notification in the *Commonwealth of Australia Gazette*, dated 24th April, 1940, concerning Denis Thorman Shortridge is cancelled.—(Ex. Min. No. 27—Approved 22nd May, 1940.)

Proceedings of the Australian Medical Boards.

VICTORIA.

A MEETING of the Medical Board of Victoria was held on May 7, 1940.

Registrations.—The following persons were registered:

Maclurkin, Iver Douglas Macarthur Brodie, L.R.C.P., L.R.C.S. (Edinburgh), L.R.F.P.S. (Glasgow), 1938.

Bamber, Leo, M.B., 1917, Ch.M., 1924 (Univ. Sydney).

Silverman, Herbert Nathan, M.B., B.S., 1937 (Univ. Melbourne).

Ogilvie, Ethel Elizabeth Mary, M.B., B.S., 1927 (Univ. London).

Deaths.—The following deaths were reported: Francis Phillip Vincent Walsh, on April 7, 1940; Francis Alexander Newman, on May 4, 1940.

Other Matters.—Several other matters received the consideration of the Board, but, as they are either *sub judice* or confidential, it is not possible to publish them.

Any medical practitioner whose address differs from that shown opposite his name in the Medical Register is advised to forward his correct address to the Secretary of the Medical Board, as the register will shortly be revised. Under Section 9 of the *Medical Act*, 1928, failure to notify change of address may lead to the removal of the name of a medical practitioner from the register.

SOUTH AUSTRALIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1919 *to* 1935, of South Australia, as duly qualified medical practitioners:

Perec, Chain, L.R.C.P. and S. (Edinburgh), 1939, L.R.F.P.S. (Glasgow), 1939, Adelaide.

Ferguson, William Frederick, M.B., B.S., 1940 (Univ. Melbourne), Mareeba Babies' Hospital, Adelaide.

Robinson, Alex McLaren, M.B., B.S., 1940 (Univ. Melbourne), Northfield Infectious Diseases Hospital, Adelaide.

The following additional qualification has been registered:

Chinner, Melville Ernest, M.B., B.S., 1925 (Univ. Adelaide), M.R.C.P. (London), 1939.

NOTICE.

A CLINICAL meeting of the medical staff of the Royal Prince Alfred Hospital, Sydney, will be held at the hospital on Monday, June 10, 1940, at 4.30 o'clock p.m. The meeting will take the form of a demonstration of the following: (*a*) two cases of division of the ureter (followed by a discussion on methods of reimplantation); (*b*) long-standing dysmenorrhœa due to endometriosis; (*c*) carcinoma of the *cervix uteri* with recurrence (microscopic sections only). All medical practitioners are invited to be present.

Books Received.

MY LIFE, by Havelock Ellis; 1940. London: William Heinemann Limited. Demy 8vo, pp. 159, with illustrations. Price: 15s. net.

THE HAPPY MOTHER AND CHILD, by I. Y. Ross, O.B.E., M.B., Ch.B., with an introduction by E. Pritchard, M.A., M.D., F.R.C.P.; 1940. Melbourne: Robertson and Mullens Limited. Crown 8vo, pp. 192, with illustrations. Price: 5s. net.

ANATOMY OF ANIMAL TYPES FOR STUDENTS OF ZOOLOGY, by E. A. Briggs, D.Sc.; Second Edition, revised and enlarged; 1940. Sydney: Angus and Robertson Limited. Demy 8vo, pp. 310, with illustrations.

ATLAS OF FIRST AID, by David Farrell, with a foreword by F. A. Maguire, C.M.G., D.S.O., V.D., M.D., F.R.C.S.; 1940. Sydney: Angus and Robertson Limited. Crown 4to, pp. 59. Price: 5s. net.

THE HISTAMINE AND INSULIN TREATMENT OF SCHIZOPHRENIA AND OTHER MENTAL DISEASES, by H. Hill, M.R.C.P.; 1940. London: Baillière, Tindall and Cox. Crown 8vo, pp. 138. Price: 6s. net.

POISONS: THEIR ISOLATION AND IDENTIFICATION, by F. Bamford, B.Sc., with a foreword by S. Smith, M.D., F.R.C.P.; 1940. London: J. and A. Churchill Limited. Demy 8vo, pp. 352, with 21 illustrations. Price: 18s. net.

DERMATOLOGIC ALLERGY: AN INTRODUCTION IN THE FORM OF A SERIES OF LECTURES, by Marion B. Sulzberger, M.D.; 1940. Baltimore: C. C. Thomas. Medium 8vo, pp. 565, with illustration. Price: $8.50 net.

Diary for the Month.

JUNE 11.—Tasmanian Branch, B.M.A.: Branch.
JUNE 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 14.—Queensland Branch, B.M.A.: Council.
JUNE 18.—New South Wales Branch, B.M.A.: Ethics Committee.
JUNE 19.—Western Australian Branch, B.M.A.: Branch.
JUNE 20.—New South Wales Branch, B.M.A.: Clinical meeting.
JUNE 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 26.—South Australian Branch, B.M.A.: Annual meeting.
JUNE 26.—Victorian Branch, B.M.A.: Council.
JUNE 27.—New South Wales Branch, B.M.A.: Branch.
JUNE 28.—Queensland Branch, B.M.A.: Council.
JUNE 28.—Tasmanian Branch, B.M.A.: Council.
JULY 2.—New South Wales Branch, B.M.A.: Council (Quarterly).
JULY 3.—Western Australian Branch, B.M.A.: Council.
JULY 3.—Victorian Branch, B.M.A.: Branch.
JULY 4.—South Australian Branch, B.M.A.: Council.
JULY 5.—Queensland Branch, B.M.A.: Branch.

Medical Appointments.

Dr. W. C. Smith has been appointed Government Medical Officer at Murwillumbah, New South Wales.

* * *

Dr. A. J. Kennedy has been appointed a Medical Referee for the purposes of *The Workers' Compensation Acts*, 1916 *to* 1939, of Queensland.

* * *

Dr. W. B. Ryan has been appointed Superintendent of the Mental Hospital at Sunbury, Victoria, pursuant to the provisions of the *Lunacy Acts* of Victoria.

* * *

Dr. C. Farran-Ridge has been appointed Superintendent of the Mental Hospital at Beechworth, Victoria, in accordance with the provisions of the *Lunacy Acts* of Victoria.

* * *

Dr. H. J. C. Hanrahan has been appointed, pursuant to the provisions of the *Quarantine Act*, 1908-1924, Deputy Quarantine Officer at Albany, Western Australia.

* * *

Dr. S. Pearlman has been appointed Honorary Ophthalmologist to the Mental Hospital at Parkside, South Australia.

* * *

Dr. J. L. Gorman has been appointed Second Assistant Medical Superintendent of the Brisbane Mental Hospital, Goodna, in accordance with the provisions of *The Public Service Acts*, 1922-1924, and *The Mental Hygiene Act of* 1938, of Queensland.

* * *

Dr. A. S. Ellis, Dr. T. Schlicht and Dr. E. M. Catarinich have been appointed Medical Officers in the Department of Mental Hygiene of Victoria, according to the provisions of the *Public Service Act*, 1928, and the *Lunacy Acts* of Victoria.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xiv-xvii.

INNISFAIL HOSPITALS BOARD, INNISFAIL, QUEENSLAND: Resident Medical Superintendent.
QUEEN'S MEMORIAL INFECTIOUS DISEASES HOSPITAL, FAIRFIELD, VICTORIA: Junior Resident Medical Officer.
SOUTH AUSTRALIAN RAILWAYS: Medical Officer.
THE EASTERN SUBURBS HOSPITAL, WAVERLEY, NEW SOUTH WALES: Assistant Physicians, Assistant Surgeon.
THE ROYAL NORTH SHORE HOSPITAL OF SYDNEY, NEW SOUTH WALES: Honorary Officers.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	Associated Medical Services Limited. All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Federated Mutual Medical Benefit Society. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	Wiluna Hospital. All Contract Practice Appointments in Western Australia.

Editorial Notices.

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All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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